Before you begin: This is a big topic, and big topics beget big slide-sets. There are natural breaks at slides 166, 276, 427, 482, and 654; I placed a break time! slide at those points to mark them.
How common is DES?
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Very. It is estimated to affect $\%$ of adults age 30-60, and $\%$ of adults age 65 and older.
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Is it a significant health problem?
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It certainly can be. Studies indicate moderate-to-severe DES impacts quality-of-life to the same degree as moderate-to-severe angina.
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Is there a gender predilection?
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Dry Eye Syndrome

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Why are women more likely to have DES?
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Why are women more likely to have DES?
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Why are women more likely to have DES?

There are a number of factors, but one of the most fundamental is hormonal—hormones are protective against DES, while tend to exacerbate it.
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Do androgens play a direct role in tear-film health?
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Why are women more likely to have DES?
There are a number of factors, but one of the most fundamental is hormonal—androgens are protective against DES, while estrogens tend to exacerbate it

Because of these hormonal effects…
…women are more likely to have DES if they are receiving estrogen replacement therapy
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What is the classic clinical scenario in which a man is undergoing androgen antagonist therapy?
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**Dry Eye Syndrome**

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The *Cornea* book is maddeningly inconsistent on this score. In text, it states there is “no racial or ethnic predisposition.”
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Dry Eye Syndrome

What roles does the tear film play in ocular health and function?
What roles does the tear film play in ocular health and function? There are three:

--?
--?
--?
What roles does the tear film play in ocular health and function? There are three:
--Facilitates diffusion of \[\text{vascular status}\] to the cornea
--?
--?
What roles does the tear film play in ocular health and function? There are three:
--Facilitates diffusion of oxygen to the avascular cornea
--?
--?
What roles does the tear film play in ocular health and function? There are three:
--Facilitates diffusion of oxygen to the avascular cornea
--Assists in clearing debris from the corneal surface
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What roles does the tear film play in ocular health and function?
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--Facilitates diffusion of oxygen to the avascular cornea
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--Provides a glassy-smooth refracting surface at the air-cornea interface (or more accurately, the air-tear film interface)
What roles does the tear film play in ocular health and function?
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Where does the tear film reside? (The answer is not ‘on the surface of the eye.’)
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Where does the tear film reside? (The answer is not ‘on the surface of the eye.’)
The bulk of tear volume is in the tear strip or lake (aka the tear meniscus) resting on the lower-lid margin
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Tear lake (strip; meniscus)
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How does the tear volume get from the tear strip up onto the ocular surface where it’s needed?
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Courtesy of the action of the two words.

Dry Eye Syndrome
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The bulk of tear volume is in the tear strip or lake (aka the tear *meniscus*) resting on the lower-lid margin

How does the tear volume get from the tear strip up onto the ocular surface where it’s needed?
Courtesy of the action of the upper lid (UL) . During a blink, the UL travels down across most of the extent of the (the lower lid goes up a little, but not much).
What roles does the tear film play in ocular health and function?

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How does the tear volume get from the tear strip up onto the ocular surface where it’s needed?
Courtesy of the action of the upper lid (UL). During a blink, the UL travels down across most of the extent of the interpalpebral fissure (the lower lid goes up a little, but not much).
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How does the tear volume get from the tear strip up onto the ocular surface where it’s needed?
Courtesy of the action of the upper lid (UL). During a blink, the UL travels down across most of the extent of the interpalpebral fissure (the lower lid goes up a little, but not much). As it goes down the UL wipes debris off the surface and into the lake. As it goes back up, the UL exerts a capillary-attraction force on the aqueous in the tear lake, thereby pulling it up across the ocular surface. (The oil layer follows along.)
Dry Eye Syndrome

The tear film is comprised of basic components.
The tear film is comprised of three basic components.
The tear film is comprised of three basic components. What are they?
--?
--?
--?
Dry Eye Syndrome

The tear film is comprised of three basic components. What are they?
--Lipid
--Aqueous
--Mucin
The tear film is comprised of three basic components. What are they?
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How are the three components physically related to one another?
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How are the three components physically related to one another?
The aqueous and mucus components are intermixed into a single, gel-like layer (the ‘
phase’), which in turn is covered by a lipid layer.
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How are the three components physically related to one another? The aqueous and mucus components are intermixed into a single, gel-like layer (the ‘mucoaqueous phase’), which in turn is covered by a lipid layer.
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How are the three components physically related to one another? The aqueous and mucus components are intermixed into a single, gel-like layer (the ‘mucoaqueous phase’), which in turn is covered by a lipid layer. This is the two-phase model of the tear film.
Two-phase model of the tear film. Schematic drawing of the structure of the tear film showing the outer lipid layer and the mucoaqueous layer.
The tear film is comprised of three basic components. What are they?
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The aqueous and mucus components are intermixed into a single, gel-like layer (the ‘mucoaqueous phase’), which in turn is covered by a lipid layer. This is the two-phase model of the tear film.

As an aside: Briefly, what is the tripartite model of the tear film?
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As an aside: Briefly, what is the tripartite model of the tear film? The idea that the tear film is composed of three separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers.
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As an aside: Briefly, what is the tripartite model of the tear film?
The idea that the tear film is composed of three separate and distinct layers each comprised of one component, i.e., separate mucus, aqueous, and lipid layers.

There is a problem with the tripartite model—what is it?
The tear film is comprised of three basic components. What are they?
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There is a problem with the tripartite model—what is it? While once widely accepted, consensus now is it’s incorrect.
The tear film is comprised of three basic components. What are they?

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The lipid component/layer makes key contributions to the stability and effectiveness of the tear film—what are they?

- ?
- ?

Which gland(s) produce the lipids constituting this layer?

The meibomian glands.

Dry Eye Syndrome

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The lipid component/layer makes key contributions to the stability and effectiveness of the tear film—what are they?

--Inhibit tear film evaporation, thereby keeping it on the eye longer
--?

Without a lipid layer, surface tension (along with gravity) would pull the tear film down the eye to the lake.

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- Reduce tear film surface tension, thereby keeping it on the eye longer

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How are the three components physically related to one another?
The aqueous and mucin components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

As an aside: Briefly, what is the tripartite model of the tear film?
The idea that the tear film is composed of three separate and distinct layers each comprised of one component, i.e., separate mucus, aqueous, and lipid layers.

There is a problem with the tripartite model—what is it?
While once widely accepted, consensus now is it’s incorrect.
The tear film is comprised of three basic components. What are they?

- Lipid
- Aqueous
- Mucin

How are the three components physically related to one another?
The aqueous and mucin components are intermixed into a single, gel-like layer (the ‘mucogel phase’), which in turn is covered by a lipid layer.

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The lipid component/layer makes key contributions to the stability and effectiveness of the tear film—what are they?

-- Inhibit tear film evaporation, thereby keeping it on the eye longer
-- Reduce tear film surface tension, thereby keeping it on the eye longer
---- Without a lipid layer, surface tension (along with gravity) would pull the tear film down the eye to the lake
-- Facilitate visual acuity by providing a smooth surface.
The tear film is comprised of three basic components. What are they?

--Lipid
--Aqueous
--Mucin

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--Facilitate visual acuity by providing a smooth refracting surface

Which gland(s) produce the lipids constituting this layer?
The meibomian glands
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Which gland(s) produce the lipids constituting this layer?
The meibomian glands are embedded within the specific structure.

The meibomian glands are embedded within the

Dry Eye Syndrome
The tear film is comprised of three basic components. What are they?

- Lipid
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- Without a lipid layer, surface tension (along with gravity) would pull the tear film down the eye to the lake
- Facilitate visual acuity by providing a smooth refracting surface

Which gland(s) produce the lipids constituting this layer?

The meibomian glands. The meibomian glands are embedded within the tarsal plates.

Upper lid, lower lid, or both?

Both.
The tear film is comprised of three basic components. What are they?

--- Lipid
--- Aqueous
--- Mucin

How are the three components physically related to one another?
The aqueous and mucin components are intermixed into a single, gel-like layer (the 'mucocanine phase'), which in turn is covered by a lipid layer.

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Which gland(s) produce the lipids constituting this layer?
The meibomian glands.

The meibomian glands are embedded within the tarsal plates.

Upper lid, lower lid, or both?
Both.
Meibomian glands

Dry Eye Syndrome
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Which gland(s) produce the lipids constituting this layer? The meibomian glands. The meibomian glands are embedded within the tarsal plates. The product of a meibomian gland is called meibum. There are up to twice as many meibomian glands in the upper lids. The meibomian glands are innervated primarily by the parasympathetic system.
The tear film is comprised of three basic components. What are they?

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Which gland(s) produce the lipids constituting this layer?
The meibomian glands.

The meibomian glands are embedded within the tarsal plates.
The product of a meibomian gland is called meibum.
There are up to twice as many meibomian glands in the upper lids.
Dry Eye Syndrome

Upper lid

Lower lid

Meibomian glands
The tear film is comprised of three basic components. What are they?

- Lipid
- Aqueous
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Dry Eye Syndrome
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What gland-type secretes the aqueous portion of the tear film?

- Aqueous

How many lacrimal glands are there (in each orbit)?

- Lots! But we think of them as being in one of two groups:
  - The main lacrimal gland
  - The accessory lacrimal glands

Are they innervated?

- Yes, primarily by nerves of the parasympathetic system

There is a problem with the tripartite model—what is it?

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What gland-type secretes the aqueous portion of the tear film?
Lacrimal gland
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What gland-type secretes the aqueous portion of the tear film? Lacrimal gland

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Are they innervated? Yes, primarily by nerves of the parasympathetic system.

Dry Eye Syndrome
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What gland-type secretes the aqueous portion of the tear film?
Lacrimal gland

How many lacrimal glands are there (in each orbit)?
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- ?
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Lacrimal gland

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Are they innervated?

Yes, primarily by nerves of the parasympathetic system.
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Lacrimal gland

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Lacrimal gland

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- The accessory lacrimal glands

Are they innervated?

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Where is the main lacrimal gland located?

The superotemporal orbit.
The tear film is comprised of three basic components. What are they?

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Are they innervated?
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Where is the main lacrimal gland located?
The superotemporal orbit.

It’s divided into two lobes—what are they called?
The orbital and palpebral lobes.
The tear film is comprised of three basic components. What are they?

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Where is the main lacrimal gland located?
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It’s divided into two lobes—what are they called?
The orbital and palpebral lobes.
The orbital lobe of the lacrimal gland ($L_o$) and the palpebral lobe of the lacrimal gland ($L_p$) are separated by the lateral horn of the levator aponeurosis (Ap) (FYI: LPS = levator palpebralis superioris; Wh = Whitnall’s ligament)
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What gland-type secretes the aqueous portion of the tear film?
Lacrimal gland

How many lacrimal glands are there (in each orbit)?
Lots! But we think of them as being in one of two groups:
--The main lacrimal gland
--The accessory lacrimal glands

There are two eponymous accessory glands—what are they?
--Glands of Krauss, found in the fornices
--Glands of Wolfring, found near the tarsal plates

Are these large, singular structures a la the main lac gland?
No, they are two sets of (much smaller) glands distributed throughout the eye.

Dry Eye Syndrome
The tear film is comprised of three basic components. What are they?
--Lipid
--Aqueous
--Mucin

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--Glands of Krauss
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There is a problem with the tripartite model—what is it?
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The tear film is comprised of three basic components: lipid, aqueous, and mucin. How are the three components physically related to one another? The aqueous and mucus components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

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What gland-type secretes the aqueous portion of the tear film? Lacrimal gland.

How many lacrimal glands are there (in each orbit)? Lots! But we think of them as being in one of two groups: the main lacrimal gland and the accessory lacrimal glands.

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What is the primary location for each?
The tear film is comprised of three basic components. What are they?
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How are the three components physically related to one another?
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Are these large, singular structures a la the main lac gland?

Dry Eye Syndrome
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What gland-type secretes the aqueous portion of the tear film?

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In addition to secreting its aqueous component, the lacrimal glands contribute important ‘microconstituents’ of the tear film. What are these?

--Electrolytes

--Solutes

--Proteins

How many lacrimal glands are there (in each orbit)?

Lots! But we think of them as being in one of two groups:

--The main lacrimal gland

--The accessory lacrimal glands

Are they innervated?

Yes, primarily by nerves of the parasympathetic system

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What is the primary role of electrolytes in the tear film?
To regulate tear-film osmolarity.

Why is tear-film osmolarity important?
Because of its associated osmotic-pressure gradient. The corneal epithelial cell membranes are freely permeable to water but not solutes; ie, they are semi-permeable. Recall the rule regarding semi-permeable membranes: Solvent follows solute. What this means is, if tear-film osmolarity gets too high, water within the epithelial cells will be pulled out of them via the resulting osmotic gradient. (This is a really important concept, peeps!)

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Because of its associated osmotic-pressure gradient. The corneal epithelial cell membranes are freely permeable to water but not solutes; i.e., they are semi-permeable. Recall the rule regarding semi-permeable membranes: Solvent follows solute. What this means is, if tear-film osmolarity gets too high, water within the epithelial cells will be pulled out of them via the resulting osmotic gradient. (This is a really important concept, peeps!)

In a sentence or two, what is osmolarity?
The concentration of solutes in a fluid—literally, the number of solute-particles in a given amount of solvent (fluid). With regard to the tear film, it is expressed in milliosmoles per liter (mOsm/L).

In DES, do you expect tear osmolarity to be higher, or lower than normal?
Higher. Think of it this way: If the tear film is inadequate—if there’s not enough fluid there—it means the solute-particles are dissolved in a smaller amount of fluid, which in turn means the concentration of the particles will be higher.
The tear film is comprised of three basic components. What are they?

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Solvent follows solute.

What this means is, if tear-film osmolarity gets too high, water within the epithelial cells will be pulled out of them via the resulting osmotic gradient. (This is a really important concept, peeps!)

In a sentence or two, what is osmolarity?
The concentration of solutes in a fluid—literally, the number of solute-particles in a given amount of solvent (fluid). With regard to the tear film, it is expressed in milliosmoles per liter (mOsm/L).
The tear film is comprised of three basic components. What are they?

- Lipid
- Aqueous
- Mucin

How are the three components physically related to one another?
The aqueous and mucus components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

As an aside: Briefly, what is the tripartite model of the tear film?
The idea that the tear film is composed of three separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers.

There is a problem with the tripartite model—what is it?
While once widely accepted, consensus now is it’s incorrect.

What gland-type secretes the aqueous portion of the tear film?
Lacrimal gland

How many lacrimal glands are there (in each orbit)?
Lots! But we think of them as being in one of two groups:
- The main lacrimal gland
- The accessory lacrimal glands

Are they innervated?
Yes, primarily by nerves of the parasympathetic system.

There are two eponymous accessory glands—what are they?

What is the primary location for each?
- Glands of Krauss, found in the fornices
- Glands of Wolfring, found near the tarsal plates

Are these large, singular structures a la the main lac gland?
No, they are two sets of (much smaller) glands distributed throughout the orbit.

In addition to secreting its aqueous component, the lacrimal glands contribute important ‘microconstituents’ of the tear film. What are these?
- Electrolytes
- Solute

What is the primary role of electrolytes in the tear film?
To regulate tear-film osmolarity.

What is the primary role of solutes in the tear film?

In a sentence or two, what is osmolarity?
The concentration of solutes in a fluid—literally, the number of solute-particles in a given amount of solvent (fluid). With regard to the tear film, it is expressed in milliosmoles per liter (mOsm/L).

In DES, do you expect tear osmolarity to be higher, or lower than normal?
Higher. Think of it this way: If the tear film is inadequate—if there’s not enough fluid there—it means the solute-particles are dissolved in a smaller amount of fluid, which in turn means the concentration of the particles will be higher.
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What gland-type secretes the aqueous portion of the tear film?
Lacrimal gland

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In addition to secreting its aqueous component, the lacrimal glands contribute important ‘microconstituents’ of the tear film. What are these?
--Electrolytes
--Solute
--Proteins

What is the primary role of electrolytes in the tear film?
To regulate tear-film osmolarity.

Why is tear-film osmolarity important?
Because of its associated osmotic-pressure gradient. The corneal epithelial cell membranes are freely permeable to water but not solutes; ie, they are semi-permeable. Recall the rule regarding semi-permeable membranes: Solvent follows solute. What this means is, if tear-film osmolarity gets too high, water within the epithelial cells will be pulled out of them via the resulting osmotic gradient. (This is a really important concept, peeps!)

Dry Eye Syndrome

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What gland-type secretes the aqueous portion of the tear film?
Lacrimal gland

How many lacrimal glands are there (in each orbit)?
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There is a problem with the tripartite model—what is it?
While once widely accepted, consensus now is it’s incorrect.

What gland-type secretes the aqueous portion of the tear film?
Lacrimal gland

How many lacrimal glands are there (in each orbit)?
Lots! But we think of them as being in one of two groups:

--The main lacrimal gland
--The accessory lacrimal glands

Are they innervated?
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In DES, do you expect tear osmolarity to be higher, or lower than normal?
Higher. Think of it this way: If the tear film is inadequate—if there’s not enough fluid there—it means the solute-particles are dissolved in a smaller amount of fluid, which in turn means the concentration of the particles will be higher.

What is normal tear osmolarity value? (It’s a range.)
296 ± 10 milliosmoles per liter (mOsm/L)

What tear-osmolarity value is widely acknowledged as indicative of at least mild DES?
308 (mOsm/L)
The tear film is comprised of three basic components. What are they?

- Lipid
- Aqueous
- Mucin

How are the three components physically related to one another?

The aqueous and mucus components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

As an aside: Briefly, what is the tripartite model of the tear film?

The idea that the tear film is composed of three separate and distinct layers each comprised of one component, i.e., separate mucus, aqueous, and lipid layers.

There is a problem with the tripartite model—what is it?

While once widely accepted, consensus now is it’s incorrect.

What gland-type secretes the aqueous portion of the tear film?

- Lacrimal gland

How many lacrimal glands are there (in each orbit)?

Lots! But we think of them as being in one of two groups:

- The main lacrimal gland
- The accessory lacrimal glands

Are they innervated?

Yes, primarily by nerves of the parasympathetic system.

There are two eponymous accessory glands—what are they?

- Glands of Krauss, found in the fornices
- Glands of Wolfring, found near the tarsal plates

Are these large, singular structures a la the main lac gland?

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In addition to secreting its aqueous component, the lacrimal glands contribute important ‘microconstituents’ of the tear film. What are these?

- Electrolytes
- Solutes
- Proteins

What is the primary role of electrolytes in the tear film?

To regulate tear-film osmolarity.

Why is tear-film osmolarity important?

Because of its associated osmotic-pressure gradient. The corneal epithelial cell membranes are freely permeable to water but not solutes; i.e., they are semi-permeable. Recall the rule regarding semi-permeable membranes: Solvent follows solute. What this means is, if tear-film osmolarity gets too high, water within the epithelial cells will be pulled out of them via the resulting osmotic gradient. (This is a really important concept, peeps!)

Dry Eye Syndrome

In a sentence or two, what is osmolarity?

The concentration of solutes in a fluid—literally, the number of solute particles in a given amount of solvent. With regard to the tear film, it is expressed in milliosmoles per liter (mOsm/L).

In DES, do you expect tear osmolarity to be higher, or lower than normal?

Higher. Think of it this way: If the tear film is inadequate—if there’s not enough fluid there—it means the solute particles are dissolved in a smaller amount of fluid, which in turn means the concentration of the particles will be higher.

What is normal tear osmolarity value? (It’s a range.)

296 ± # milliosmoles per liter (mOsm/L)

What tear-osmolarity value is widely acknowledged as indicative of at least mild DES?

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- Aqueous
- Mucin

How are the three components physically related to one another?
The aqueous and mucus components are intermixed into a single, gel-like layer (the ‘mucoaqueous phase’), which in turn is covered by a lipid layer.

As an aside: Briefly, what is the tripartite model of the tear film?
The idea that the tear film is composed of three separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers.

There is a problem with the tripartite model—what is it?
While once widely accepted, consensus now is it’s incorrect.

What gland-type secretes the aqueous portion of the tear film?
Lacrimal gland

How many lacrimal glands are there (in each orbit)?
Lots! But we think of them as being in one of two groups:

- The main lacrimal gland
- The accessory lacrimal glands

Are they innervated?
Yes, primarily by nerves of the parasympathetic system.

There are two eponymous accessory glands—what are they?
- Glands of Krauss, found in the fornices
- Glands of Wolfring, found near the tarsal plates

Are these large, singular structures a la the main lac gland?
No, they are two sets of (much smaller) glands distributed throughout the orbit.

What is the primary location for each?

- Glands of Krauss, found in the fornices
- Glands of Wolfring, found near the tarsal plates

In addition to secreting its aqueous component, the lacrimal glands contribute important ‘microconstituents’ of the tear film. What are these?
- Electrolytes
- Solutes
- Proteins

What is the primary role of electrolytes in the tear film?
To regulate tear-film osmolarity.

What is normal tear osmolarity value? (It’s a range.)
296 ± 10 milliosmoles per liter (mOsm/L)

In DES, do you expect tear osmolarity to be higher, or lower than normal?
Higher. Think of it this way: If the tear film is inadequate—if there’s not enough fluid there—it means the solute-particles are dissolved in a smaller amount of fluid, which in turn means the concentration of the particles will be higher.

In a sentence or two, what is osmolarity?
The concentration of solutes in a fluid—literally, the number of solute-particles in a given amount of solvent. With regard to the tear film, it is expressed in milliosmoles per liter (mOsm/L).

What tear-osmolarity value is widely acknowledged as indicative of at least mild DES?
308 (mOsm/L)
The tear film is comprised of three basic components: lipid, aqueous, and mucin. These components are physically related in that the aqueous and mucin components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

In addition to secreting its aqueous component, the lacrimal glands contribute important ‘microconstituents’ of the tear film. What are these? Electrolytes and solutes.

What is the primary role of electrolytes in the tear film? To regulate tear-film osmolarity.

In a sentence or two, what is osmolarity? The concentration of solutes in a fluid—literally, the number of solute-particles in a given amount of solvent (fluid). With regard to the tear film, it is expressed in milliosmoles per liter (mOsm/L).

What is normal tear osmolarity value? (It’s a range.)
296 ± 10 mOsm/L

What tear-osmolarity value is widely acknowledged as indicative of at least mild DES? 308 mOsm/L.

In DES, do you expect tear osmolarity to be higher or lower than normal? Higher. Think of it this way: If the tear film is inadequate—if there isn’t enough fluid there—it means the solute-particles are dissolved in a smaller amount of fluid, which in turn means the concentration of the particles will be higher.

While once widely accepted, consensus now is it’s incorrect to think of the tear film as tripartite. The tripartite model suggests the tear film is composed of three separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers. However, there is a problem with the tripartite model—what is it? While once widely accepted, consensus now is it’s incorrect.
The tear film is comprised of three basic components. What are they?

- Lipid
- Aqueous
- Mucin

How are the three components physically related to one another?

The aqueous and mucus components are intermixed into a single, gel-like layer (the ‘mucoaqueous phase’), which in turn is covered by a lipid layer.

As an aside: Briefly, what is the tripartite model of the tear film?

The idea that the tear film is composed of three separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers.

There is a problem with the tripartite model—what is it?

While once widely accepted, consensus now is it’s incorrect.

What gland-type secretes the aqueous portion of the tear film?

Lacrimal gland

How many lacrimal glands are there (in each orbit)?

Lots! But we think of them as being in one of two groups:

- The main lacrimal gland
- The accessory lacrimal glands

Are they innervated?

Yes, primarily by nerves of the parasympathetic system.

There are two eponymous accessory glands—what are they?

- Glands of Krauss, found in the fornices
- Glands of Wolfring, found near the tarsal plates

Are these large, singular structures a la the main lac gland?

No, they are two sets of (much smaller) glands distributed throughout the orbit.

In addition to secreting its aqueous component, the lacrimal glands contribute important ‘microconstituents’ of the tear film. What are these?

- Electrolytes
- Solutes
- Proteins

What is the primary role of electrolytes in the tear film?

To regulate tear-film osmolarity.

Why is tear-film osmolarity important?

Because of its associated osmotic-pressure gradient. The corneal epithelial cell membranes are freely permeable to water but not solutes; ie, they are semi-permeable. Recall the rule regarding semi-permeable membranes: Solvent follows solute. What this means is, if tear-film osmolarity gets too high, water within the epithelial cells will be pulled out of them via the resulting osmotic gradient. (This is a really important concept, peeps!)

What is normal tear osmolarity value? (It’s a range.)

296 ± 10

What tear-osmolarity value is widely acknowledged as indicative of at least mild DES?

308*

*This is from EyeWiki, not the BCSC. Caveat emptor.
The tear film is comprised of three basic components. What are they?

- Lipid
- Aqueous
- Mucin

How are the three components physically related to one another? The aqueous and mucin components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

As an aside: Briefly, what is the tripartite model of the tear film? The idea that the tear film is composed of three separate and distinct layers each comprised of one component, i.e., separate mucus, aqueous, and lipid layers.

There is a problem with the tripartite model—what is it? While once widely accepted, consensus now is it’s incorrect.

What gland-type secretes the aqueous portion of the tear film? The lacrimal gland.

How many lacrimal glands are there (in each orbit)? Lots! But we think of them as being in one of two groups:

- The main lacrimal gland
- The accessory lacrimal glands

Are they innervated? Yes, primarily by nerves of the parasympathetic system.

There are two eponymous accessory glands—what are they? Glands of Krauss, found in the fornices, Glands of Wolfring, found near the tarsal plates.

Are these large, singular structures a la the main lac gland? No, they are two sets of (much smaller) glands distributed throughout the orbit.

In addition to secreting its aqueous component, the lacrimal glands contribute important ‘microconstituents’ of the tear film. What are these? Electrolytes, Solutes, Proteins.

What is the primary role of electrolytes in the tear film? To regulate tear-film osmolarity.

Why is tear-film osmolarity important?

There is a problem with the tripartite model—what is it? While once widely accepted, consensus now is it’s incorrect.
The tear film is comprised of three basic components. What are they?

- **Lipid**
- **Aqueous**
- **Mucin**

How are the three components physically related to one another?
The aqueous and mucus components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

As an aside: Briefly, what is the tripartite model of the tear film?
The idea that the tear film is composed of three separate and distinct layers each comprised of one component, i.e., separate mucus, aqueous, and lipid layers.

There is a problem with the tripartite model—what is it?
While once widely accepted, consensus now is it’s incorrect.

What gland-type secretes the aqueous portion of the tear film?

- **Lacrimal gland**

How many lacrimal glands are there (in each orbit)?

- Lots! But we think of them as being in one of two groups:
  - The main lacrimal gland
  - The accessory lacrimal glands

Are they innervated?
Yes, primarily by nerves of the parasympathetic system.

What is the primary location for each?

- Glands of Krauss, found in the fornices
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Are these large, singular structures a la the main lac gland?
No, they are two sets of (much smaller) glands distributed throughout the orbit.

In addition to secreting its aqueous component, the lacrimal glands contribute important ‘microconstituents’ of the tear film. What are these?

- **Electrolytes**
- **Solutes**
- **Proteins**

What is the primary role of electrolytes in the tear film?
To regulate tear-film osmolarity.

Why is tear-film osmolarity important?
Because of its associated two-words gradient.

There is a problem with the tripartite model—what is it?
While once widely accepted, consensus now is it’s incorrect.
The tear film is comprised of three basic components: lipid, aqueous, and mucin.

How are the three components physically related to one another?

The aqueous and mucus components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

As an aside: Briefly, what is the tripartite model of the tear film?

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There is a problem with the tripartite model—what is it?

While once widely accepted, consensus now is it's incorrect.

What gland-type secretes the aqueous portion of the tear film?

Lacrimal gland

How many lacrimal glands are there (in each orbit)?

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Are they innervated?

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What is the primary role of electrolytes in the tear film?

To regulate tear-film osmolarity.

Why is tear-film osmolarity important?

Because of its associated osmotic-pressure gradient.

What is Dry Eye Syndrome?
The tear film is comprised of three basic components. What are they?

- Lipid
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- Electrolytes
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- Proteins

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Why is tear-film osmolarity important?
Because of its associated osmotic-pressure gradient. The corneal epi cell membranes are freely permeable to water but not solutes; i.e., they are semi-permeable. Recall the rule regarding semi-permeable membranes: solvent follows solute. What this means is, if tear-film osmolarity gets too high, water within the epi cells will be pulled out of them via the resulting osmotic gradient. (This is a really important concept, peeps!)

Dry Eye Syndrome
The tear film is comprised of three basic components. What are they?

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There is a problem with the tripartite model—what is it?

While once widely accepted, consensus now is it’s incorrect.
The tear film is comprised of three basic components. What are they?

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While once widely accepted, consensus now is it’s incorrect.
The tear film is comprised of three basic components. What are they?
--Lipid
--Aqueous
--Mucin

How are the three components physically related to one another?
The aqueous and mucus components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

As an aside: Briefly, what is the tripartite model of the tear film?
The idea that the tear film is composed of three separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers.

There is a problem with the tripartite model—what is it?
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While once widely accepted, consensus now is it’s incorrect.

What gland-type secretes the aqueous portion of the tear film?
- Lacrimal gland

How many lacrimal glands are there (in each orbit)?
- Lots! But we think of them as being in one of two groups:
  - The main lacrimal gland
  - The accessory lacrimal glands

Are they innervated?
- Yes, primarily by nerves of the parasympathetic system.

In addition to secreting its aqueous component, the lacrimal glands contribute important ‘microconstituents’ of the tear film. What are these?
- Electrolytes
- Solutes
- Proteins

What is the primary protein on the tear film?
- Immunoglobulin, specifically IgA

Is it just hanging out in the tear film, or does it contribute to local host-defenses?
- It is an important defense component.
The tear film is comprised of three basic components. What are they?

- Lipid
- Aqueous
- Mucin

How are the three components physically related to one another?
The aqueous and mucin components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

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What gland-type secretes the aqueous portion of the tear film?
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--Electrolytes
--Solute
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What is the primary protein on the tear film? Immunoglobulin, specifically IgA

Is it just hanging out in the tear film, or does it contribute to local host-defenses?

Yes, primarily IgA immunoglobulin.

Dry Eye Syndrome
The tear film is comprised of three basic components. What are they?

- Lipid
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How are the three components physically related to one another?
The aqueous and mucus components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

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What is the primary protein on the tear film?
Immunoglobulin, specifically IgA.

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The tear film is comprised of three basic components. What are they?

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What is the chief function of the mucin component of the mucoaqueous layer?

As an aside: Briefly, what is the tripartite model of the tear film?

The idea that the tear film is composed of three separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers

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While once widely accepted, consensus now is it’s incorrect
The tear film is comprised of three basic components. What are they? --Lipid --Aqueous --Mucin

How are the three components physically related to one another?
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While once widely accepted, consensus now is it’s incorrect.

What is the chief function of the mucin component of the mucoaqueous layer? 
Facilitating surface wetting.

Dry Eye Syndrome
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There is a problem with the tripartite model—what is it?
While once widely accepted, consensus now is it’s incorrect.

What is the chief function of the mucin component of the mucoaqueous layer?
Facilitating surface wetting by transforming the epithelial surface from a hydrophobic to a hydrophilic state.

Dry Eye Syndrome
The tear film is comprised of three basic components. What are they? --Lipid
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How are the three components physically related to one another? The aqueous and mucus components are intermixed into a single, gel-like layer (the 'mucoaqueous phase'), which in turn is covered by a lipid layer.

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What is the chief function of the mucin component of the mucoaqueous layer? Facilitating surface wetting by transforming the epithelial surface from a hydrophobic to a hydrophilic state.

Which cells are the chief producers of mucins?
The tear film is comprised of three basic components. What are they?
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What is the chief function of the mucin component of the mucoaqueous layer? Facilitating surface wetting by transforming the epithelial surface from a hydrophobic to a hydrophilic state

Which cells are the chief producers of mucins? Goblet cells

As an aside: Briefly, what is the tripartite model of the tear film? The idea that the tear film is composed of three separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers

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What is the chief function of the mucin component of the mucoaqueous layer?
Facilitating surface wetting by transforming the epithelial surface from a hydrophobic to a hydrophilic state

Which cells are the chief producers of mucins?
Goblet cells, which are found in the epithelium

As an aside: Briefly, what is the tripartite model of the tear film?
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The aqueous and mucus components are intermixed into a single, gel-like layer (the ‘mucoaqueous phase’), which in turn is covered by a lipid layer.

As an aside: Briefly, what is the tripartite model of the tear film?
The idea that the tear film is composed of three separate and distinct layers each comprised of one component, ie, separate mucus, aqueous, and lipid layers.

What is the chief function of the mucin component of the mucoaqueous layer?
Facilitating surface wetting by transforming the epithelial surface from a hydrophobic to a hydrophilic state.

Which cells are the chief producers of mucins?
Goblet cells, which are found in the conjunctival epithelium.

There is a problem with the tripartite model—what is it?
While once widely accepted, consensus now is it’s incorrect.
We saw this depiction of the two-phase model of the tear film earlier in the set… But are now ready to note the presence and location of mucin.
We saw this depiction of the *two-phase model of the tear film* earlier in the set... But are now ready to note the presence and location of mucin. Note that in addition to the ‘soluble’ mucins of the mucoaqueous layer, there are ‘membrane-bound’ mucins contributing to the structure of the corneal epithelium.
We saw this depiction of the *two-phase model of the tear film* earlier in the set… But are now ready to note the presence and location of mucin. Note that in addition to the ‘soluble’ mucins of the mucoaqueous layer, there are ‘membrane-bound’ mucins contributing to the *glycocalyx* of the corneal epithelium.
We saw this depiction of the two-phase model of the tear film earlier in the set… But are now ready to note the presence and location of mucin. Note that in addition to the ‘soluble’ mucins of the mucoaqueous layer, there are ‘membrane-bound’ mucins contributing to the glycocalyx of the corneal epithelium.

For more on the tear film, see slide-set K47
Next we will look at the **Lacrimal Functional Unit (LFU)** and its role in tear production and maintenance.

We saw this depiction of the *two-phase model of the tear film* earlier in the set… But are now ready to note the presence and location of mucin. Note that in addition to the ‘soluble’ mucins of the mucocutaneous layer, there are ‘membrane-bound’ mucins contributing to the *glycocalyx* of the corneal epithelium.
What is the lacrimal functional unit (LFU)?
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The LFU is the complex, integrated system responsible for the regulation, production, and health of the tear film.
**What is the lacrimal functional unit (LFU)?**

The LFU is the complex, integrated system responsible for the regulation, production, and health of the tear film. Think of it as the reflex arc responsible for the production of the components of the tear film.
Recall that a reflex arc has three components: A sensory limb consisting of sensory receptors and afferent nerves.

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Recall that a reflex arc has three components: A sensory limb consisting of sensory receptors and afferent nerves, a motor limb consisting of efferent nerves and the effector end-organ.

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**What is the lacrimal functional unit (LFU)?**

The LFU is the complex, integrated system responsible for the regulation, production, and health of the tear film. Think of it as the reflex arc responsible for the production of the components of the tear film.
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In the LFU, the sensory limb consists of ocular-surface nociceptors connected to branches of two nerves.

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**In the LFU**, the sensory limb consists of ocular-surface nociceptors connected to branches of V1 and V2.

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Recall that a reflex arc has three components: A sensory limb consisting of sensory receptors and afferent nerves, a motor limb consisting of efferent nerves and the effector end-organ, and a CNS integration center that connects the afferent and efferent limbs.

In the LFU, the sensory limb consists of ocular-surface nociceptors connected to branches of V1 and V2. The motor limb consisting of the lacrimal, meibomian, and goblet glands/cells (innervated by...)

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**In the LFU**, the sensory limb consists of ocular-surface nociceptors connected to branches of $V_1$ and $V_2$. The motor limb consisting of the lacrimal, meibomian, and goblet glands/cells (innervated by parasympathetics) as well as the **Orbicularis oculi** muscle (innervated by $CN#$).

**What is the lacrimal functional unit (LFU)?**
The LFU is the complex, integrated system responsible for the regulation, production, and health of the tear film. Think of it as **the reflex arc responsible for the production of the components of the tear film.**
Dry Eye Syndrome

**The LFU**

- **Sensory limb**
  - Sensory receptors: Ocular-surface nociceptors
  - Afferent nerves: Branches of V₁ and V₂

- **CNS integration center**

- **Motor limb**
  - Efferent nerves: --P’sympathetics, --CN7
  - Effectors: --Glands, ----Lacrimal, ----M’bomian, ----Goblet, --Orbicularis

Recall that a reflex arc has three components: A *sensory limb* consisting of sensory receptors and afferent nerves, a *motor limb* consisting of efferent nerves and the effector end-organ, and a *CNS integration center* that connects the afferent and efferent limbs.

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**What is the lacrimal functional unit (LFU)?**

The LFU is the complex, integrated system responsible for the regulation, production, and health of the tear film. Think of it as the reflex arc responsible for the production of the components of the tear film.
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**In the LFU**, the sensory limb consists of ocular-surface nociceptors connected to branches of V1 and V2. The motor limb consisting of the lacrimal, meibomian, and goblet glands/cells (innervated by parasympathetics) as well as the orbicularis oculi muscle (innervated by CN7). CNS integration takes place in the brainstem.

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In the LFU, the sensory limb consists of ocular-surface nociceptors connected to branches of V1 and V2. The motor limb consisting of the lacrimal, meibomian, and goblet glands/cells (innervated by parasympathetics) as well as the orbicularis oculi muscle (innervated by CN7). CNS integration takes place in the brainstem.

**What is the lacrimal functional unit (LFU)?**
The LFU is the complex, integrated system responsible for the regulation, production, and health of the tear film. Think of it *the reflex arc responsible for the production of the components of the tear film.*
Dry Eye Syndrome

The LFU
CNS integration center

Sensory limb
Motor limb

Sensory receptors
Afferent nerves
Brainstem
Efferent nerves

Ocular-surface nociceptors
Branches of V1 and V2
Brainstem
--P’sympathetics

Effector end-organ

Recall that a reflex arc has three components: A sensory limb consisting of sensory receptors and afferent nerves, a motor limb consisting of efferent nerves and the effector end-organ, and a CNS integration center that connects the afferent and efferent limbs.

In the LFU, the sensory limb consists of ocular-surface nociceptors connected to branches of V1 and V2. The motor limb consisting of the lacrimal, meibomian, and goblet glands/cells (innervated by parasympathetics) as well as the orbicularis oculi muscle (innervated by CN7). CNS integration takes place in the brainstem.

What is the lacrimal functional unit (LFU)?
The LFU is the complex, integrated system responsible for the regulation, production, and health of the tear film. Think of it the reflex arc responsible for the production of the components of the tear film.

For more on the LFU, see slide-set K46
(This is a good point in the set to take a break)
We are ready (finally!) to tackle the pathophysiology of DES…
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

We are ready (finally!) to tackle the pathophysiology of DES… Which commences with something the importance of which was stressed earlier in the slide-set.
The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

We are ready (finally!) to tackle the pathophysiology of DES… Which commences with something the importance of which was stressed earlier in the slide-set.
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity**.

(Reiterating for emphasis)

*What are the units of measurement for tear-film osmolarity?*
The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

(Reiterating for emphasis)

What are the units of measurement for tear-film osmolarity?
milli-osmols per liter (mOsm/L)
The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

(Reiterating for emphasis)

What are the units of measurement for tear-film osmolarity? milli-osmols per liter (mOsm/L)

What is the osmolarity of the normal tear film?
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

(Reiterating for emphasis)

What are the units of measurement for tear-film osmolarity?
milli-osmols per liter (mOsm/L)

What is the osmolarity of the normal tear film?
Around 290-300 mOsm/L
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity**.

*(Reiterating for emphasis)*

**What are the units of measurement for tear-film osmolarity?**
milli-osmols per liter (mOsm/L)

**What is the osmolarity of the normal tear film?**
Around 290-300 mOsm/L

**How high does tear osmolarity have to get to be clinically significant?**
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

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milli-osmols per liter (mOsm/L)

What is the osmolarity of the normal tear film?
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How high does tear osmolarity have to get to be clinically significant?
308 (per EyeWiki)
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

In what two fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?

1) ?

2) ?

or...

Tear hyperosmolarity
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

In what two fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?

1) The amount of aqueous can be inadequate to maintain normal osmolarity.
2) The amount of aqueous can be too high to maintain normal osmolarity.
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

In what two fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?

1) The amount of aqueous produced can be inadequate to maintain normal osmolarity.  2) The amount of aqueous lost can be too high to maintain normal osmolarity.

Tear hyperosmolarity
The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity**.

In what two fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?

1) The amount of aqueous produced can be inadequate to maintain normal osmolarity. This state is known as...  
2) The amount of aqueous lost can be too high to maintain normal osmolarity.
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

In what two fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?

1) The amount of aqueous produced can be inadequate to maintain normal osmolarity. This state is known as...

   **Aqueous Tear Deficiency**

2) The amount of aqueous lost can be too high to maintain normal osmolarity.
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

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Aqueous Tear Deficiency

2) The amount of aqueous lost can be too high to maintain normal osmolarity. This state is known as...

?  

Tear hyperosmolarity
The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

In what two fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?

1) The amount of aqueous produced can be inadequate to maintain normal osmolarity. This state is known as Aqueous Tear Deficiency.

2) The amount of aqueous lost can be too high to maintain normal osmolarity. This state is known as Evaporative Dry Eye.

Tear hyperosmolarity
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

In what two fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?

Head’s up: Later in the set we’re gonna add a third mechanism leading to tear hyperosmolarity.

Aqueous Tear Deficiency

Evaporative Dry Eye

Tear hyperosmolarity
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

While it’s a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:

- Problem with the **aqueous component**
  - Aqueous Tear Deficiency

Tear hyperosmolarity
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

While it's a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:

- Problem with the aqueous component
  - Aqueous Tear Deficiency
  - Tear hyperosmolarity

Evaporative Dry Eye
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

While it’s a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:

- Problem with the **aqueous component**: Aqueous Tear Deficiency
- Problem with the **component**: Evaporative Dry Eye

Tear hyperosmolarity
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

While it’s a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:

- **Problem with the aqueous component**
  - Aqueous Tear Deficiency

- **Problem with the lipid component**
  - Evaporative Dry Eye

Tear hyperosmolarity
The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

Let’s drill down on both, starting with ATD.

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Tear hyperosmolarity
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

*What are the three classic tests of aqueous tear production?*

*Wait for it…*

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Wait for it…OK, now answer

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What are the dimensions of the test strips used?  
30 x 5 mm

How are the strips placed?  
With length hooked over the lid margin and the other length hanging over the front of the lid.
### What are the three classic tests of aqueous tear production?

**What does each assess? How is each performed? How is each interpreted?**

<table>
<thead>
<tr>
<th>Test name</th>
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<tr>
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</tr>
<tr>
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<td></td>
<td></td>
</tr>
<tr>
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- **Basal secretion test:** Instill anesthetic, blot, place strip, measure saturation at 5 min. Less than 3 mm wetting after 5 min = ATD
- **Schirmer I:** Basal and reflex secretion. Same, but without instilling anesthetic. Less than 5 mm wetting after 5 min = ATD
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(No question—summary slide for review)
What are the three classic tests of aqueous tear production? What does each assess? How is each performed? How is each interpreted?

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<tr>
<td>Interpretation</td>
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What are the dimensions of the test strips used? 30 x 5 mm

How are the strips placed? With 5 mm hooked over the lid margin and the other 25 mm hanging over the front of the lid

Where along the lid margin should the strip be placed? At the junction of the outer third and middle third of the lid
**Dry Eye Syndrome**

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We talking upper lid, or lower?

(Dry Eye Syndrome)

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(No question—summary slide for review)
Dry Eye Syndrome

ATD is subdivided into two categories--what are they?

- eponym
- non-eponym

Aqueous Tear Deficiency

Tear hyperosmolarity

Evaporative Dry Eye
Dry Eye Syndrome

ATD is subdivided into two categories--what are they?

Sjögren’s

Non-Sjögren’s

Aqueous Tear Deficiency

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(Statement of fact—not a question. Keep going.)
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1. **Aqueous Tear Deficiency**
2. **Dry Eye Syndrome**
3. **Tear hyperosmolarity**
4. **Evaporative Dry Eye**

What does reflex block mean?

Recall that tear production is considered largely reflexive. Thus, any break in the LFU reflex circuit will lead to ATD.

What are some of the common mechanisms producing afferent limb block?

- The most common culprits are conditions leading to corneal hypoesthesia, including:
  - Neurotrophic cornea
  - Corneal surgery
  - Post-herpetic neuropathy
  - Contact-lens wear

What are some of the common mechanisms producing efferent limb block?

- Anything that compromises CN7
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**Here’s a no-frills version of the LFU for reference**

```
                     Receptors
                        |
               Ocular surface nociceptors
                        |
                     Brainstem integration
                        |
              Cranial nerve 5
                        |
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                        |
              Cranial nerve 7
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What are some of the common mechanisms producing afferent limb block?
--The most common culprits are conditions leading to:
  two words

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**Here’s a no-frills version of the LFU for reference**

**Non-Sjögren’s**

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Receptors

Ocular surface nociceptors

Cranial nerve 5

Brainstem integration

Cranial nerve 7

Lacrimal apparatus

Receptors

Afferent limb

Nucleus

Efferent limb

Effector
In SS, aqueous hyposecretion (and therefore ATD) results from autoimmune-mediated lymphocytic infiltration of the lacrimal glands. In non-Sjögren's ATD, four broad categories of conditions leading to lacrimal gland hyposecretion have been identified. What are they?

- Aqueous Tear Deficiency
- Dry Eye Syndrome
- Tear hyperosmolarity
- Non-Sjögren's Lacrimal deficiency
- Lacrimal duct obstruction
- Reflex block
- Systemic drug effect

What does reflex block mean? Recall that tear production is considered largely reflexive. Thus, any break in the LFU reflex circuit will lead to ATD.

What are some of the common mechanisms producing afferent limb block?
-- The most common culprits are conditions leading to corneal hypoesthesia, including:
---- Neurotrophic cornea
---- Corneal surgery
---- Post-herpetic neuropathy
---- ?

Here's a no-frills version of the LFU for reference

- Ocular surface nociceptors
- Cranial nerve 5
- Brainstem integration
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  - Contact-lens wear

**What are some of the common mechanisms producing efferent limb block?**
- Anything that compromises CN7

Here’s a no-frills version of the LFU for reference:

- **Receptors**
- **Afferent limb**
- **Nucleus**
- **Efferent limb**
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**Ocular surface nociceptors**

**Cranial nerve 5**

**Brainstem integration**

**Cranial nerve 7**

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</thead>
<tbody>
<tr>
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Sjögren’s Non-Sjögren’s

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Dry Eye Syndrome

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Non-Sjögren’s

- Aqueous Tear Deficiency
- Tear hyperosmolarity
- Evaporative Dry Eye

Sjögren’s

Systemic drug effect

Of the 100 best-selling drugs in the US, how many list dry eye as a side effect? Twenty-two!
Dry Eye Syndrome

- Non-Sjögren’s
  - Aqueous Tear Deficiency
  - Tear hyperosmolarity

- Sjögren’s

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- Reflex block

Systemic drug effect

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Dry Eye Syndrome

Three very general classes of pharmacologic effect are implicated in inducing DES—what are they?

- Anti-histamines
- Anti-depressants
- Anti-hypertensives
- Anti-emetics
- Anti-Parkinson’s
- Anti-psychotics
- Anti-adrenergics
- Oral contraceptive pills

Systemic drug effect

- Aqueous Tear Deficiency
- Evaporative Dry Eye
- Tear hyperosmolarity

Non-Sjögren’s

- Lacrimal deficiency
- Lacrimal duct obstruction
- Reflex block

Sjögren’s
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

Three very general classes of pharmacologic effect are implicated in inducing DES—what are they?

- Anti-histamine effect
- Anti-cholinergic effect
- Hormonal effect

Lacrimal deficiency  Lacrimal duct obstruction  Reflex block
Sjögren’s  Non-Sjögren’s  Systemic drug effect
Aqueous Tear Deficiency  Evaporative Dry Eye
Tear hyperosmolality
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Aqueous Tear Deficiency

Evaporative Dry Eye

Systemic drug effect

Sjögren’s

Non-Sjögren’s

Lacrimal deficiency

Lacrimal duct obstruction

Reflex block

Tear hyperosmolarity
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Aqueous Tear Deficiency

Sjögren's

Evaporative Dry Eye

Tear hyperosmosmolarity

Systemic drug effect

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Systemic drug effect

Sjögren’s

Non-Sjögren’s

Aqueous Tear Deficiency

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Tear hyperosmolarity

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Evaporative Dry Eye

Tear hyperosmolarity

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Non-Sjögren's Sjögren's

Lacrimal deficiency Lacrimal duct obstruction Reflex block

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**Systemic drug effect**

- Anti-histamine effect
- Anti-cholinergic effect
- Hormonal effect

**Non-Sjögren’s**

- Aqueous Tear Deficiency
- Evaporative Dry Eye
- Tear hyperosmolarity

**Sjögren’s**

- Lacrimal deficiency
- Lacrimal duct obstruction
- Reflex block

**Drug Effect**

- Lacrimal deficiency
- Lacrimal duct obstruction
- Reflex block
Dry Eye Syndrome

50 Ways to Take a Break

(This is a good point in the set to take a break)
Dry Eye Syndrome

Evaporative dry eye is subdivided into two categories—what are they?

Sjögren’s

Non-Sjögren’s

Aqueous Tear Deficiency

Tear hyperosmolarity

Evaporative Dry Eye

word

←that word’s antonym
Evaporative dry eye is subdivided into two categories—what are they?

- **Intrinsic**
  - Evaporative Dry Eye

- **Extrinsic**
  - Non-Sjögren’s
  - Aqueous Tear Deficiency

**Dry Eye Syndrome**
In this context, to what do the terms intrinsic and extrinsic refer?
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In this context, to what do the terms *intrinsic* and *extrinsic* refer? *Intrinsic* evaporative dry eye refers to any cause related to the eyelids. *Extrinsic* refers to any non-eyelid factor that promoted evaporation.

What are the three main etiologies of *intrinsic* evaporative dry eye?

- Sjögren’s
- Non-Sjögren’s
- Aqueous Tear Deficiency
- Evaporative Dry Eye
- Tear hyperosmolarity
In this context, to what do the terms intrinsic and extrinsic refer? *Intrinsic* evaporative dry eye refers to any cause related to the eyelids. *Extrinsic* refers to any non-eyelid factor that promoted evaporation.

What are the three main etiologies of *intrinsic* evaporative dry eye?

- Meibomian gland dysfunction (MGD)
- Widened lid fissure
- Reduced blink rate

**Sjögren’s**

**Non-Sjögren’s**

**Aqueous Tear Deficiency**

**Tear hyperosmolarity**

**Evaporative Dry Eye**

**Intrinsic**

**Extrinsic**
Dry Eye Syndrome

MGD demonstrates a racial predilection—what group has a notably higher prevalence?

Meibomian gland dysfunction (MGD) 
- Widened lid fissure
- Reduced blink rate

Sjögren’s Non-Sjögren’s

Aqueous Tear Deficiency

Intrinsic

Evaporative Dry Eye

Tear hyperosmolarity
MGD demonstrates a racial predilection—what group has a notably higher prevalence? Asians

-Meibomian gland dysfunction (MGD)
  -Widened lid fissure
  -Reduced blink rate

Intrinsic

Evaporative Dry Eye

Tear hyperosmolarity

Sjögren’s

Non-Sjögren’s

Aqueous Tear Deficiency

Tear
In one simple word, what is the underlying issue in most cases of MGD?

MGD demonstrates a racial predilection—what group has a notably higher prevalence? Asians
**Dry Eye Syndrome**

*MGD demonstrates a racial predilection—what group has a notably higher prevalence?*  
Asians

*In one simple word, what is the underlying issue in most cases of MGD?*  
of gland output leading to inadequate volume of tear-film meibum

---

**Meibomian gland dysfunction (MGD)**

- Widened lid fissure
- Reduced blink rate

**Intrinsic**

**Extrinsic**

- Sjögren’s
- Non-Sjögren’s
- Aqueous Tear Deficiency
- Evaporative Dry Eye

**Tear hyperosmolarity**
MGD demonstrates a racial predilection—what group has a notably higher prevalence? Asians

In one simple word, what is the underlying issue in most cases of MGD? 
**Obstruction** of gland output leading to inadequate volume of tear-film meibum

---

**Disease**

- *Dry Eye Syndrome*
  - *Intrinsic* 
  - *Extrinsic*

- *Non-Sjögren’s*
- *Sjögren’s*
- *Aqueous Tear Deficiency*

- *Evaporative Dry Eye*

**Obstruction**

- *Meibomian gland dysfunction (MGD)*
- *Widened lid fissure*
- *Reduced blink rate*

**Factors**

- *Tear hyperosmolarity*
In one simple word, what is the underlying issue in most cases of MGD? **Obstruction** of gland output leading to inadequate volume of tear-film meibum.

Obstructive MGD is divided into two subtypes—what are they?

- **Intrinsic**
  - Meibomian gland dysfunction (MGD)
  - Widened lid fissure
  - Reduced blink rate

- **Extrinsic**
  - Evaporative Dry Eye
  - Tear hyperosmolarity

MGD demonstrates a racial predilection—what group has a notably higher prevalence? **Asians**
Dry Eye Syndrome

*MGD demonstrates a racial predilection—what group has a notably higher prevalence? Asians*

*In one simple word, what is the underlying issue in most cases of MGD? Obstruction of gland output leading to inadequate volume of tear-film meibum*

Obstructive *MGD is divided into two subtypes—what are they?* Cicatrising and noncicatrising

**Obstructive MGD**

- Widened lid fissure
- Reduced blink rate

**Intrinsic**

- Meibomian gland dysfunction (MGD)

**Extrinsic**

**Aqueous Tear Deficiency**

- Sjögren’s
- Non-Sjögren’s

**Evaporative Dry Eye**

**Tear hyperosmolarity**
Dry Eye Syndrome

MGD demonstrates a racial predilection—what group has a notably higher prevalence? Asians

In one simple word, what is the underlying issue in most cases of MGD? Obstruction of gland output leading to inadequate volume of tear-film meibum

Obstructive MGD is divided into two subtypes—what are they? Cicatrizing and noncicatrizing

Meibomian gland dysfunction (MGD) is a cause of cicatrizing obstructive MGD owing to meibomian gland dysfunction (MGD), which leads to obstructions of gland output leading to inadequate volume of tear-film meibum.

The Cornea book highlights three causes of cicatrizing obstructive MGD—not what are they?
---?
---?
---?

The Cornea book highlights three causes of noncicatrizing obstructive MGD—what are they?
- Rosacea
- Seborrheic dermatitis
- Atopy (I know—weird that it appears on both lists)
**Dry Eye Syndrome**

**MGD** demonstrates a racial predilection—what group has a notably higher prevalence? Asians

In one simple word, what is the underlying issue in most cases of MGD? **Obstruction** of gland output leading to inadequate volume of tear-film meibum

Obstructive MGD is divided into two subtypes—what are they? **Cicatrizing** and noncicatrizing

**Meibomian gland dysfunction (MGD)**

- Widened lid fissure
- Reduced blink rate

**Evaporative**

- Reduced tear volume
- Tear hyperosmolarity

**Tear hyperosmolarity**

The Cornea book highlights three causes of cicatrizing obstructive MGD—what are they?

- Trachoma
- Mucous-membrane pemphigoid
- Atopy

**Extrinsic**

- Non-Sjögren's
- Sjögren's

**Intrinsic**

- Aqueous tear deficiency
- Meibomian gland dysfunction (MGD)

**Tear deficiency**

- Reduced tear volume
- Increased tear evaporation

**Widened lid fissure**

- Reduced blink rate
- Tear film instability

**Reduction in tear osmolarity**

- Tear hyperosmolarity
- Reduced tear volume
In one simple word, what is the underlying issue in most cases of MGD? Obstruction of gland output leading to inadequate volume of tear-film meibum.

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The Cornea book highlights three causes of cicatrizing obstructive MGD—what are they?
--Trachoma
--Mucous-membrane pemphigoid (aka ocular cicatricial pemphigoid)
--Atopy

Asians

Meibomian gland dysfunction (MGD)

Widened lid fissure

Reduced blink rate

Etiology

Extrinsic

Vaporative

Dry Eye

Tear hyperosmolarity
Dry Eye Syndrome

MGD demonstrates a racial predilection—what group has a notably higher prevalence? Asians

In one simple word, what is the underlying issue in most cases of MGD? **Obstruction** of gland output leading to inadequate volume of tear-film meibum

Obstructive MGD is divided into two subtypes—what are they? **Cicatrizing** and noncicatrizing

*Meibomian gland dysfunction* (MGD) can present in two forms:
- Widened lid fissure
- Reduced blink rate

The Cornea book highlights three causes of cicatrizing obstructive MGD—what are they?
- Trachoma
- Mucous-membrane pemphigoid (*aka* ocular cicatricial pemphigoid)
- Atopy

Extrinsic

Dry Eye

Extrinsic

Evaporative

Tear hyperosmolarity

Intrinsic

Aqueous tear deficiency

Widened lid fissure

Meibomian gland dysfunction (MGD)

Reduced blink rate
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In one simple word, what is the underlying issue in most cases of MGD? Obstruction of gland output leading to inadequate volume of tear-film meibum

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Meibomian gland dysfunction (MGD)  Widened lid fissure  Reduced blink rate

Extrinsic

Evaporative

Dry Eye

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--Atopy

The book highlights three causes of noncicatrizing obstructive MGD—what are they?
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Dry Eye Syndrome

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**Meibomian gland dysfunction (MGD)**

**Widened lid fissure**

**Reduced blink rate**

Extrinsic

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Dry Eye

**Tear hyperosmolarity**

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--Seborrheic dermatitis
--Atopy
Dry Eye Syndrome

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--Rosacea
--Seborrheic dermatitis
--Atopy

Not a typo (on my part)—the Cornea book lists atopy under both causes

Tear hyperosmolarity
**Dry Eye Syndrome**

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**Cicatriz ing** and noncicatriz ing

Meibomian gland        Widened
Reduced

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--Rosacea
--Seborrheic dermatitis
--Atopy

In a nutshell, what is rosacea?
A chronic skin condition often involving the eyelids

What is the cause?
It is unknown at this time

Is there a gender predilection? A racial predilection? Age predilection?
Yes, women are more likely to be affected. No. Middle-aged.

What are the classic nonocular findings on exam?
--Midface erythema
--Pustules/papules
--Thickening of nasal skin (called rhinophyma)

What does the Cornea book call "the mainstay of therapy" for rosacea?
Oral tetracyclines
Dry Eye Syndrome

MGD demonstrates a racial predilection—what group has a notably higher prevalence? Asians.

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Rosacea acute vs chronic.
Dry Eye Syndrome

MGD demonstrates a racial predilection—what group has a notably higher prevalence?
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In one simple word, what is the underlying issue in most cases of MGD?
Obstruction of gland output leading to inadequate volume of tear-film meibum

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Cicatriziing and noncicatriziing

Meibomian gland

Reduced blink rate

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Obstruction of gland output leading to inadequate volume of tear-film meibum

Obstructive MGD is divided into two subtypes—what are they?

Cicatrizing and noncicatrizing

Meibomian gland

Widened

Reduced blink rate

Extrinsic

Evaporative

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What is the cause?

It is unknown at this time

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Dry Eye Syndrome

**MGD demonstrates a racial predilection**—what group has a notably higher prevalence? Asians

**In one simple word, what is the underlying issue in most cases of MGD?**
**Obstruction** of gland output leading to inadequate volume of tear-film meibum

**Obstructive MGD is divided into two subtypes**—what are they? **Cicatrizing** and noncicatrizing

In a nutshell, what is rosacea?
A chronic skin condition often involving the eyelids

What is the cause?
It is unknown at this time

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--Mucous-membrane pemphigoid (aka ocular cicatricial pemphigoid)
--Atopy

The book highlights three causes of noncicatrizing obstructive MGD—what are they?
--Rosacea
--Seborrheic dermatitis
--Atopy

**Extrinsic**

**Intrinsic**

**Non-Sjögren's**

**Sjögren's**

**Evaporative Dry Eye**

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**Widened lid fissure**

**Meibomian gland dysfunction (MGD)**

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Meibomian gland Widened

Reduced blink rate

Extrinsic

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*Is there a gender predilection?* Yes, are more likely to be affected. No. Middle-aged.

*What are the classic nonocular findings on exam?* --Midface erythema --Pustules/papules --Thickening of nasal skin (called rhinophyma)

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**Meibomian gland**

Widened

Reduced

Blink rate

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Dry Eye

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In a nutshell, what is **rosacea**?

A chronic skin condition often involving the eyelids

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**What is the cause?**

It is unknown at this time

---

Is there a gender predilection?

Yes, women are more likely to be affected

---

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MVsF
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- Widened
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Extrinsic

- Foreign body
- Infections
- Allergies
- Trauma
- Vascular disorders
- Drugs
- Surgery

Intrinsic

- Aqueous tear deficiency
- Evaporative dry eye

Non-Sjögren’s

- Reduced tear production
- Gland dysfunction

Sjögren’s

- Reduced tear production
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**Dry Eye Syndrome**

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Can young individuals get rosacea?
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Meibomian gland

Reduced blink rate

Extrinsic

Aqueous Tear Deficiency

Widened lid fissure

Meibomian gland dysfunction (MGD)

Tear hyperosmolarity

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Rosacea: Midface erythema
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Rosacea

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Consumption of alcohol (honorable mention if you said consumption of spicy food
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Next finding
Dry Eye Syndrome

Rosacea: Papules/pustules
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**MGD demonstrates a racial predilection**—what group has a notably higher prevalence? Asians

**In one simple word, what is the underlying issue in most cases of MGD?**

**Obstruction** of gland output leading to inadequate volume of tear-film meibum

**Obstructive MGD is divided into two subtypes**—what are they?

**Cicatrizng** and noncicatrizng

---

**In a nutshell, what is rosacea?**

A chronic skin condition often involving the eyelids

**What is the cause?**

It is unknown at this time

**Is there a gender predilection? A racial predilection? Age predilection?**

Yes, ♀ are more likely to be affected. No. Middle-aged.

**What are the classic nonocular findings on exam?**

--Midface erythema
--Pustules/papules
--Thickening of nasal skin (called rhinophyma)
Dry Eye Syndrome

**MGD demonstrates a racial predilection**—what group has a notably higher prevalence?
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*In one simple word, what is the underlying issue in most cases of MGD?*
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---

**Meibomian gland**

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What are the classic nonocular findings on exam?
--Midface erythema
--Pustules/papules
--Thickening of nasal skin (called *rhinophyma*)

---

Extrinsic

Reduced blink rate

**Corneal**

---

Widened

Rosacea

--Seborrheic dermatitis

Atopy

---

Cicatrising

Trachoma

Mucous-membrane pemphigoid (aka *ocular cicatricial pemphigoid*)

Atopy

---

Non-cicatrising

Rosacea

---

---
Dry Eye Syndrome

Rosacea: Rhinophyma
Dry Eye Syndrome

MGD demonstrates a racial predilection—what group has a notably higher prevalence? Asians

In one simple word, what is the underlying issue in most cases of MGD? Obstruction of gland output leading to inadequate volume of tear-film meibum

Obstructive MGD is divided into two subtypes—what are they? Cicatrizng and noncicatrizng

- Meibomian gland
- Widened
- Reduced
- Blink rate
- Extrinsic
- Evaporative
- Dry Eye
- Aqueous tear deficiency
- Widened lid fissure
- Meibomian gland dysfunction (MGD)

In a nutshell, what is rosacea? A chronic skin condition often involving the eyelids

What is the cause? It is unknown at this time

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Yes, ♀ are more likely to be affected. No. Middle-aged.

What are the classic nonocular findings on exam?
- Midface erythema
- Pustules/papules
- Thickening of nasal skin (called rhinophyma)

What does the Cornea book call “the mainstay of therapy” for rosacea?
**Dry Eye Syndrome**

*MGD demonstrates a racial predilection*—what group has a notably higher prevalence?

Asians

*In one simple word, what is the underlying issue in most cases of MGD?*

**Obstruction** of gland output leading to inadequate volume of tear-film meibum

*Obstructive MGD is divided into two subtypes*—what are they?

**Cicatrizing** and noncicatrizing

**Meibomian gland**

- Widened
- Reduced blink rate

**Extrinsic**

**Non-Sjögren's**

**Sjögren's**

*In a nutshell, what is rosacea?*

A chronic skin condition often involving the eyelids

**What is the cause?**

It is unknown at this time

**Is there a gender predilection?** *A racial predilection? Age predilection?*

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- Pustules/papules
- Thickening of nasal skin (called *rhinophyma*)

**What does the Cornea book call “the mainstay of therapy” for rosacea?**

Oral
Dry Eye Syndrome

MGD demonstrates a racial predilection—what group has a notably higher prevalence? Asians

In one simple word, what is the underlying issue in most cases of MGD? Obstruction of gland output leading to inadequate volume of tear-film meibum

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In a nutshell, what is rosacea? A chronic skin condition often involving the eyelids

What is the cause? It is unknown at this time

Is there a gender predilection? A racial predilection? Age predilection? Yes, ♀ are more likely to be affected. No. Middle-aged.

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What does the Cornea book call “the mainstay of therapy” for rosacea? Oral tetracyclines
What are the causes of a widened lid fissure?
--?
--?
--?

- Meibomian gland dysfunction (MGD)
- Reduced blink rate

- Sjögren’s
- Non-Sjögren’s

- Aqueous Tear Deficiency
- Evaporative Dry Eye

- Tear hyperosmolarity
What are the causes of a widened lid fissure?
--Forward displacement of the globe (ie, proptosis/exophthalmos)
--?
--?

Dry Eye Syndrome

Aqueous Tear Deficiency
Sjögren’s
Non-Sjögren’s

Tear hyperosmolarity

Evaporative Dry Eye

Reduced blink rate

Extrinsic

Meibomian gland dysfunction (MGD)

Intrinsic

Widened lid fissure
What are the causes of a widened lid fissure?
--Forward displacement of the globe (i.e., proptosis/exophthalmos)
--Increased innervation to the lid retractors such as occurs in [three words]
--?
Dry Eye Syndrome

What are the causes of a widened lid fissure?
--Forward displacement of the globe (ie, proptosis/exophthalmos)
--Increased innervation to the lid retractors such as occurs in thyroid eye disease
--?

Meibomian gland dysfunction (MGD)

Reduced blink rate

Intrinsic

Extrinsic

Widened lid fissure

Evaporative Dry Eye

Tear hyperosmolarity

Sjögren’s

Non-Sjögren’s

Aqueous Tear Deficiency
What are the causes of a widened lid fissure?
-- Forward displacement of the globe (i.e., proptosis/exophthalmos)
-- Increased innervation to the lid retractors such as occurs in thyroid eye disease
-- Congenital craniofacial malformations resulting in shallow orbits

**Dry Eye Syndrome**

- Meibomian gland dysfunction (MGD)
- Reduced blink rate

**Widened lid fissure**

**Intrinsic**

- Sjögren’s
  - Aqueous Tear Deficiency
  - Tear hyperosmolarity

**Extrinsic**

- Non-Sjögren’s
- Evaporative Dry Eye
What are the causes of a widened lid fissure?
--Forward displacement of the globe (i.e., proptosis/exophthalmos)
--Increased innervation to the lid retractors such as occurs in thyroid eye disease
--Congenital craniofacial malformations resulting in shallow orbits
Dry Eye Syndrome

What group of congenital craniofacial malformations are strongly associated with shallow orbits?

- Increased innervation to the lid retractors such as occurs in thyroid eye disease
- Congenital craniofacial malformations resulting in shallow orbits

What are the causes of a widened lid fissure?

- Forward displacement of the globe (i.e., proptosis/exophthalmos)
- Congenital craniofacial malformations resulting in shallow orbits

Meibomian gland dysfunction (MGD)

Reduced blink rate

Sjögren’s Non-Sjögren’s

Aqueous Tear Deficiency

Intrinsic Extrinsic

Widened lid fissure

Evaporative Dry Eye

Tear hyperosmolarity
What group of congenital craniofacial malformations are strongly associated with shallow orbits? The craniosynostoses.
What group of congenital craniofacial malformations are strongly associated with shallow orbits?
The craniosynostoses

In a nutshell, what is the causal mechanism of the craniosynostoses?
Premature closure of one or more cranial sutures

What are the four craniosynostoses discussed in detail in the BCSC?

- Sjögren’s
- Non-Sjögren’s
- Aqueous Tear Deficiency
- Evaporative Dry Eye

What are the causes of a widened lid fissure?
- Forward displacement of the globe (ie, proptosis/exophthalmos)
- Increased innervation to the lid retractors such as occurs in thyroid eye disease
- Congenital craniofacial malformations resulting in shallow orbits

Tear hyperosmolarity

Reduced blink rate

Widened lid fissure

Meibomian gland dysfunction (MGD)
What group of congenital craniofacial malformations are strongly associated with shallow orbits?

The craniosynostoses

In a nutshell, what is the causal mechanism of the craniosynostoses?

Premature closure of one or more cranial sutures

does not apply

What are the causes of a widened lid fissure?

- Forward displacement of the globe (ie, proptosis/exophthalmos)
- Increased innervation to the lid retractors such as occurs in thyroid eye disease
- Congenital craniofacial malformations resulting in shallow orbits

Tear hyperosmolarity

What are the four craniosynostoses discussed in detail in the BCSC? two words

- The craniosynostoses
- Sjögren’s
- Non-Sjögren’s
- Aqueous Tear Deficiency

Evaporative Dry Eye

Intrinsic

Reduced blink rate

Widened lid fissure

Meibomian gland dysfunction (MGD)
What are the causes of a widened lid fissure?

- Forward displacement of the globe (i.e., proptosis/exophthalmos)
- Increased innervation to the lid retractors such as occurs in thyroid eye disease
- Congenital craniofacial malformations resulting in shallow orbits

What group of congenital craniofacial malformations are strongly associated with shallow orbits? The craniosynostoses

In a nutshell, what is the causal mechanism of the craniosynostoses? Premature closure of one or more cranial sutures

Dry Eye Syndrome

Meibomian gland dysfunction (MGD)

Widened lid fissure

Reduced blink rate

Intrinsic

Extrinsic

Sjögren’s

Non-Sjögren’s

Aqueous Tear Deficiency

Evaporative Dry Eye

Tear hyperosmolarity
What group of congenital craniofacial malformations are strongly associated with shallow orbits?

The craniosynostoses

- ?
- ?
- ?
- ?

What are the four craniosynostoses discussed in detail in the BCSC?

In a nutshell, what is the causal mechanism of the craniosynostoses?

Premature closure of one or more cranial sutures

What are the causes of a widened lid fissure?

- Forward displacement of the globe (i.e., proptosis/exophthalmos)
- Increased innervation to the lid retractors such as occurs in thyroid eye disease
- Congenital craniofacial malformations resulting in shallow orbits

Tear hyperosmolarity

Intrinsic

Extrinsic

Sjögren’s

Non-Sjögren’s

Aqueous Tear Deficiency

Evaporative Dry Eye

Reduced blink rate

Meibomian gland dysfunction (MGD)
What group of congenital craniofacial malformations are strongly associated with shallow orbits?

**The craniosynostoses**

- Crouzon
- Apert
- Pfeiffer
- Saethre-Chotzen

What are the four craniosynostoses discussed in detail in the BCSC?

In a nutshell, what is the causal mechanism of the craniosynostoses?

Premature closure of one or more cranial sutures

What causes a widened lid fissure?

- Forward displacement of the globe (i.e., proptosis/exophthalmos)
- Increased innervation to the lid retractors such as occurs in thyroid eye disease
- Congenital craniofacial malformations resulting in shallow orbits

What is the causal mechanism of tear hyperosmolarity?

What are the causes of a widened lid fissure?

- Forward displacement of the globe (i.e., proptosis/exophthalmos)
- Increased innervation to the lid retractors such as occurs in thyroid eye disease
- Congenital craniofacial malformations resulting in shallow orbits

Intrinsic

Extrinsic

Dry Eye Syndrome

Sjögren’s

Non-Sjögren’s

Aqueous Tear Deficiency

Evaporative Dry Eye

Tear hyperosmolarity
What group of congenital craniofacial malformations are strongly associated with shallow orbits?
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- Crouzon
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In a nutshell, what is the causal mechanism of the craniosynostoses?
Premature closure of one or more cranial sutures

What are the four craniosynostoses discussed in detail in the BCSC?
- Crouzon
- Apert
- Pfeiffer
- Saethre-Chotzen

Of these,  is/are associated with shallow orbits

Sjögren’s
Non-Sjögren’s
Aqueous Tear Deficiency

Intrinsic
Extrinsic
Evaporative Dry Eye
Tear hyperosmolarity

Reduced blink rate
Dry Eye syndrome
Intrinsic
Extrinsic
Non-Sjögren’s
Sjögren’s
Meibomian gland dysfunction (MGD)
What group of congenital craniofacial malformations are strongly associated with shallow orbits?

The craniosynostoses

- Crouzon
- Apert
- Pfeiffer
- Saethre-Chotzen

In a nutshell, what is the causal mechanism of the craniosynostoses?

Premature closure of one or more cranial sutures

What are the four craniosynostoses discussed in detail in the BCSC?

Of these, three is/are associated with shallow orbits

Which one?

Sjögren’s

Non-Sjögren’s

Aqueous Tear Deficiency

Intrinsic

Evaporative Dry Eye

Reduced blink rate

Tear hyperosmolarity
Dry Eye Syndrome

Meibomian gland dysfunction (MGD)
Reduced blink rate
Dry Eye Syndrome
Intrinsic
Extrinsic
Non-Sjögren’s
Sjögren’s
Evaporative
Dry Eye
Aqueous Tear Deficiency

What are the causes of a widened lid fissure?
- Forward displacement of the globe (ie, proptosis/exophthalmos)
- Increased innervation to the lid retractors such as occurs in thyroid eye disease
- Congenital craniofacial malformations resulting in shallow orbits

Tear hyperosmolarity

What group of congenital craniofacial malformations are strongly associated with shallow orbits?
The craniosynostoses
- Crouzon?
- Apert?
- Pfeiffer?
- Saethre-Chotzen?

In a nutshell, what is the causal mechanism of the craniosynostoses?
Premature closure of one or more cranial sutures

What are the four craniosynostoses discussed in detail in the BCSC?
Of these, three is/are associated with shallow orbits and the other isn’t. Which one?
Dry Eye Syndrome

Meibomian gland dysfunction (MGD)
Reduced blink rate

Dry Eye Syndrome

Intrinsic
Extrinsic
Non-Sjögren's
Sjögren's

Evaporative
Dry Eye

Aqueous Tear Deficiency

Tear hyperosmolarity

What are the causes of a widened lid fissure?
- Forward displacement of the globe (ie, proptosis/exophthalmos)
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What group of congenital craniofacial malformations are strongly associated with shallow orbits?
The craniosynostoses
- Crouzon
- Apert
- Pfeiffer
- Saethre-Chotzen

In a nutshell, what is the causal mechanism of the craniosynostoses?
Premature closure of one or more cranial sutures

What are the four craniosynostoses discussed in detail in the BCSC?
Of these, three is/are associated with shallow orbits and the other isn’t. Which one?
For more on the craniosynostoses, see slide-set P22
Dry Eye Syndrome

Causes of reduced blink rate can be divided into two categories--what are they?

Reduced blink rate

Intrinsic
- Meibomian gland dysfunction (MGD)
- Widened lid fissure

Extrinsic

Sjögren’s
- Aqueous Tear Deficiency

Non-Sjögren’s

Dry Eye
- Tear hyperosmolarity
Dry Eye Syndrome

Causes of reduced blink rate can be divided into two categories--what are they?
Physiological (ie, a normal phenomenon), and pathological

Reduced blink rate

Intrinsic

Meibomian gland dysfunction (MGD)
Widened lid fissure

Extrinsic

Sjögren’s
Non-Sjögren’s

Aqueous Tear Deficiency

Evaporative Dry Eye

Tear hyperosmolarity
Dry Eye Syndrome

Causes of reduced blink rate can be divided into two categories--what are they?
Physiological (ie, a normal phenomenon), and pathological

What is the most common physiological cause of reduced blink rate?

Reduced blink rate

Intrinsic

Evaporative Dry Eye

Sjögren’s

Non-Sjögren’s

Aqueous Tear Deficiency

Reduced blink rate

Meibomian gland dysfunction (MGD)

Widened lid fissure

Tear hyperosmolarity
Dry Eye Syndrome

Causes of reduced blink rate can be divided into two categories--what are they? Physiological (ie, a normal phenomenon), and pathological

What is the most common physiological cause of reduced blink rate? Sustained participation in a visually intensive task (eg, reading; computer work)
Dry Eye Syndrome

Causes of reduced blink rate can be divided into two categories—what are they? Physiological (ie, a normal phenomenon), and pathological

What is the most common physiological cause of reduced blink rate? Sustained participation in a visually intensive task (eg, reading; computer work)

What is the most common pathological cause of reduced blink rate?
Dry Eye Syndrome

*Causes of reduced blink rate can be divided into two categories--what are they? Physiological (ie, a normal phenomenon), and pathological*

*What is the most common physiological cause of reduced blink rate? Sustained participation in a visually intensive task (eg, reading; computer work)*

*What is the most common pathological cause of reduced blink rate? Parkinson’s dz*

- **Meibomian gland dysfunction (MGD)**
- **Reduced blink rate**
- **Intrinsic**
- **Evaporative Dry Eye**
- **Extrinsic**
- **Tear hyperosmolarity**

- **Sjögren’s**
- **Non-Sjögren’s**

- **Aqueous Tear Deficiency**
The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

Recall that earlier in the set we alluded to a *third* means by which tear-film status could produce hyperosmolarity and dry eye. The time to address this has arrived!

**Head’s up:** Later in the set we’re gonna add a *third* mechanism leading to tear hyperosmolarity.

In what two fundamental ways could the status of the aqueous component of the tear film lead to tear hyperosmolarity?

- Aqueous Tear Deficiency
- Evaporative Dry Eye

Tear hyperosmolarity
The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

In what other fundamental way could the status of the tear film lead to tear hyperosmolarity?
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

In what other fundamental way could the status of the tear film lead to tear hyperosmolarity?

--The tear film can too quickly, exposing the ocular surface.

Aqueous Tear Deficiency

Evaporative Dry Eye

Tear hyperosmolarity
The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

In what other fundamental way could the status of the tear film lead to tear hyperosmolarity?

--The tear film can break up too quickly, exposing the ocular surface.

Aqueous Tear Deficiency  Evaporative Dry Eye

Tear hyperosmolarity
The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity**.

*In what other fundamental way could the status of the tear film lead to tear hyperosmolarity?*

--The tear film can break up too quickly, exposing the ocular surface. This state is known as one of...

```
Aqueous Tear Deficiency ? Evaporative Dry Eye

Tear hyperosmolarity
```
The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity**.

In what other fundamental way could the status of the tear film lead to tear hyperosmolarity?

--The tear film can break up too quickly, exposing the ocular surface. This state is known as one of...

---

**Aqueous Tear Deficiency**  
**Tear Film Instability**  
**Evaporative Dry Eye**

---

Tear hyperosmolarity
The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity**.

Recalling our answers to this issue previously:

While it’s a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:

- **Problem with the aqueous component**
  - Aqueous Tear Deficiency

- **Problem with the lipid component**
  - Evaporative Dry Eye

Tear hyperosmolality
The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity**.

Recalling our answers to **this** issue previously:

*What is the answer vis a vis tear-film instability?*

*While it’s a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:*

- **Problem with the aqueous component**: Aqueous Tear Deficiency
- **Problem with the mucin component**: Tear Film Instability
- **Problem with the lipid component**: Evaporative Dry Eye

→ Tear hyperosmolarity
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of **Tear Hyperosmolarity**.

Recalling our answers to **this** issue previously:

*What is the answer vis a vis tear-film instability?*

*While it’s a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:*

- **Problem with the aqueous component**
  - Aqueous Tear Deficiency
- **Problem with the mucin component**
  - Tear Film Instability
- **Problem with the lipid component**
  - Evaporative Dry Eye

**Tear hyperosmolarity**
While it’s a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:

- **Problem with the aqueous component**
  - Aqueous Tear Deficiency

- **Problem with the mucin component**
  - Tear Film Instability

- **Problem with the lipid component**
  - Evaporative Dry Eye

**Tear hyperosmolarity**
Dry Eye Syndrome

The pathophysiology for DES damage starts with derangement of the tear film in the form of Tear Hyperosmolarity.

One important oversimplification to note is the implication below that tear-film instability is a function only of the mucin component, when in fact the status of the lipid component makes a significant contribution to tear-film (in)stability as well.

While it’s a bit of an oversimplification, we can associate the components of the tear film with the pathologic states underlying DES:

- **Problem with the aqueous component**: Aqueous Tear Deficiency
- **Problem with the mucin component (l lipid too)**: Tear Film Instability
- **Problem with the lipid component**: Evaporative Dry Eye

Tear hyperosmolarity
How is tear-film instability quantified, ie, what clinical exam maneuver is used to measure it?

The tear-film break-up time (TBUT or TFBUT) assessment

How is TBUT assessed, ie, what are the steps involved?

A little fluorescein is instilled, and the pt is asked to hold their eyes open after blinking a couple of times. The tear film is observed with the cobalt-blue filter in place, and the length of time that passes until a dry spot appears is noted.

A TBUT of less than how long is considered abnormal?

10 seconds
How is tear-film instability quantified, ie, what clinical exam maneuver is used to measure it?
The tear-film break-up time (TBUT or TFBUT) assessment

Aqueous Tear Deficiency

Tear Film Instability

Tear hyperosmolarity
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Aqueous Tear Deficiency  Tear Film Instability  Evaporative Dry Eye

Tear hyperosmolarity
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The tear-film break-up time (TBUT or TFBUT) assessment

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- Aqueous Tear Deficiency
- Tear Film Instability
- Evaporative Dry Eye
- Tear hyperosmolarity
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The tear-film break-up time (TBUT or TFBUT) assessment

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Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity
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The tear-film break-up time (TBUT or TFBUT) assessment.

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How is tear-film instability quantified, ie, what clinical exam maneuver is used to measure it?
The tear-film break-up time (TBUT or TFBUT) assessment

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A TBUT of less than how long is considered abnormal?
10 seconds
How is tear-film instability quantified, ie, what clinical exam maneuver is used to measure it? The tear-film break-up time (TBUT or TFBUT) assessment.

How is TBUT assessed, ie, what are the steps involved? A little fluorescein is instilled, and the pt is asked to hold their eyes open after blinking a couple of times. The tear film is observed with the cobalt-blue filter in place, and the length of time that passes until a dry spot appears is noted.

A TBUT of less than how long is considered abnormal? 10 seconds.

I assume Fluress drops are the way to go? No. The Cornea book states such is "not recommended" because 1) too much fluorescein gets instilled; and 2) they contain an anesthetic, which could influence the results. (In fact, TBUT assessment should occur prior to instillation of any drops.)
How is tear-film instability quantified, ie, what clinical exam maneuver is used to measure it?
The tear-film break-up time (TBUT or TFBUT) assessment

How is TBUT assessed, ie, what are the steps involved?
A little fluorescein is instilled, and the pt is asked to hold their eyes open after blinking a couple of times. The tear film is observed with the cobalt-blue filter in place, and the length of time that passes until a dry spot appears is noted.

I assume Fluress drops are the way to go?
You’d think so, but no. The Cornea book states using them is “not recommended” because 1) too much or little fluorescein gets instilled.
How is tear-film instability quantified, ie, what clinical exam maneuver is used to measure it?
The tear-film break-up time (TBUT or TFBUT) assessment.

How is TBUT assessed, ie, what are the steps involved?
A little fluorescein is instilled, and the pt is asked to hold their eyes open after blinking a couple of times. The tear film is observed with the cobalt-blue filter in place, and the length of time that passes until a dry spot appears is noted.

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How is TBUT assessed, ie, what are the steps involved?

A little fluorescein is instilled, and the pt is asked to hold their eyes open after blinking a couple of times. The tear film is observed with the cobalt-blue filter in place, and the length of time that passes until a dry spot appears is noted.

**A little fluorescein is instilled**

I assume Fluress drops are the way to go?

You’d think so, but no. The Cornea book states using them is “not recommended” because 1) too much fluorescein gets instilled; and 2) they contain an anesthetic, which could influence the results.

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**Dry Eye Syndrome**

- Aqueous Tear Deficiency
- Tear Film Instability
- Evaporative Dry Eye

Tear hyperosmolarity
Dry Eye Syndrome

How is tear-film instability quantified, ie, what clinical exam maneuver is used to measure it?
The tear-film break-up time (TBUT or TFBUT) assessment

How is TBUT assessed, ie, what are the steps involved?
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Three categories of conditions leading to TFI have been identified—what are they?
Three categories of conditions leading to TFI have been identified—what are they?
What nutritional deficiency is the leading cause of xerophthalmia worldwide?

Hypovitaminosis A

Hypovitaminosis A xerosis of the ocular surface produces what classic sign?

Bitôt spot — a foamy, white/gray area on the interpalpebral conjunctiva

What bacteria is implicated in Bitôt spot formation?

Corynebacterium xerosis

With what conditions is xerophthalmia associated in the US?

-- Dietary deficiencies
-- Chronic alcoholism
What nutritional deficiency is the leading cause of xerophthalmia worldwide? Hypovitaminosis A

Xerophthalmia

- Aqueous Tear Deficiency
- Tear Film Instability
  - Topical preservatives
  - Ocular allergy
  - Evaporative Dry Eye

Tear hyperosmolarity

Dietary deficiencies
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**Hypovitaminosis A**

How is hypovitaminosis A diagnosed?

Via serum vitamin A levels

Is hypovitaminosis A a serious condition?

Yes! The mortality rate is about 50%
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Bitôt spots: Conj lesion temporal to the cornea, shows typical dry/foamy appearance
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**Press your xerosis pts on these issues!**
What nutritional deficiency is the leading cause of xerophthalmia worldwide? Hypovitaminosis A

With what genetic disease manifesting in childhood is xerophthalmia associated? Cystic fibrosis

With what conditions is xerophthalmia associated in the US? -- Dietary deficiencies -- Chronic alcoholism

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**Wiggity what? Why would pts with a disease hallmarked by lung abnormalities be at risk for hypovitaminosis A?**
Recall that CF is also associated with pancreatic insufficiency, and thereby with Vit A malabsorption. Undiagnosed CF infants may present with xerophthalmia severe enough to produce a PUK-like picture with associated hypopyon!

With what conditions is xerophthalmia associated in the US? 
--- Dietary deficiencies 
--- Chronic alcoholism

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Dry Eye Syndrome

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**Dry Eye Syndrome**

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Is hypervitaminosis A a thing, ie, a clinically important condition? It is indeed. There is a condition of significant ophthalmic consequence—one with which you are likely familiar—that has a strong association with hypervitaminosis A. What is it? Idiopathic* intracranial hypertension (aka pseudotumor cerebri)

There is a classic (albeit far-fetched) dietary scenario associated with the development of pseudotumor cerebri—what is it? Consumption of polar bear liver
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*It is not clear to me whether expert consensus would hold that Vit A-induced intracranial hypertension would be considered idiopathic, or secondary
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Consumption of three words

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Tear Film Instability

Dry Eye

Tear hyperosmolarity
**Dry Eye Syndrome**

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**Tear Film Instability**

**Evaporative Dry Eye**

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Dry Eye Syndrome

How do preservatives in ophthalmic preparations lead to TFI?

Sjögren’s → Aqueous Tear Deficiency
Non-Sjögren’s → Evaporative Dry Eye

Tear Film Instability

Topical preservatives

Ocular allergy

Tear hyperosmolarity
How do preservatives in ophthalmic preparations lead to TFI? By provoking an inflammatory response in the conj epithelium, which in turn promotes cell apoptosis.

Dry Eye Syndrome

Sjögren’s 
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How do preservatives in ophthalmic preparations lead to TFI?
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Dry Eye Syndrome

How do preservatives in ophthalmic preparations lead to TFI?
By provoking an inflammatory response in the conj epithelium, which in turn promotes goblet cell apoptosis.

Is there a preservative that is especially notorious for doing this?

Tear Film Instability

Topical preservatives

Sjögren’s
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Xerophthalmia
How do preservatives in ophthalmic preparations lead to TFI? By provoking an inflammatory response in the conj epithelium, which in turn promotes goblet cell apoptosis.

Is there a preservative that is especially notorious for doing this? Benzalkonium chloride (aka BAK or BAC)
How does an ocular allergic condition produce TFI?

- Sjögren’s
- Non-Sjögren’s
  - Aqueous Tear Deficiency
  - Xerophthalmia
  - Topical preservatives

Tear Film Instability

- Evaporative Dry Eye
- Tear hyperosmolarity
How does an ocular allergic condition produce TFI?

Allergen antigens on the ocular surface initiate an IgE-mediated inflammatory cascade, leading to goblet-cell loss.
Dry Eye Syndrome

50 Ways to Take a Break

(This is a good point in the set to take a break)
Now that we understand how ATD, TFI and EDE lead to tear hyperosmolarity...

Aqueous Tear Deficiency  \(\rightarrow\)  Tear Film Instability  \(\rightarrow\)  Evaporative Dry Eye  \(\rightarrow\)  Tear hyperosmolarity
Now that we understand how ATD, TFI and EDE lead to tear hyperosmolarity…
Let’s examine how tear hyperosmolarity leads to DES
What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?
What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?
Hyperosmolar stress of surface epithelium
What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?

Hyperosmolar stress of surface epithelium, which significantly damages it.
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Hyperosmolar stress of surface epithelium, which significantly damages it.

Are we talking corneal epi, or conj epi?
What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?

**Hyperosmolar stress of surface epithelium**, which significantly damages it.

*Are we talking corneal epi, or conj epi?*

Both Surface epithelium damage

→ Hyperosmolary stress

→ Aqueous Tear Deficiency

→ Tear Film Instability

→ Evaporative Dry Eye

→ Tear hyperosmolarity
Dry Eye Syndrome

What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?

Hyperosmolar stress of surface epithelium, which significantly damages it.

Are we talking corneal epi, or conj epi? Both.

How might conj-epi damage directly impact TFI (and thus DES)?

DES feedback loop!

Surface epithelium damage

Hyperosmolar stress

Tear Film Instability

Tear hyperosmolality

Aqueous Tear Deficiency

Evaporative Dry Eye
What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in Dry Eye Syndrome (DES)?

Hyperosmolar stress of surface epithelium, which significantly damages it:

Are we talking corneal epi, or conjunctival epi? Both could be involved.

How might conjunctival epi (conj-epi) damage directly impact Tear Film Instability (TFI) (and thus DES)?

Recall that conjunctival goblet cells are the source of mucin, a deficit of which contributes to TFI and thus DES.

DES feedback loop!

Aqueous Tear Deficiency

Hyperosmolar stress

Tear Film Instability

Tear hyperosmolarity

Evaporative Dry Eye
What effect does tear-film hyperosmolarity produce that starts the cascade of events resulting in DES?

Hyperosmolar stress of surface epithelium, which significantly damages it

Are we talking corneal epi, or conj epi?
Both

Surface epithelium damage

Hyperosmolar stress

How might conj-epi damage directly impact TFI (and thus DES)?
Recall that conj goblet cells are the source of mucin, a deficit of which contributes to TFI and thus DES

DES feedback loop!
Evaporative Dry Eye

Dry Eye Syndrome

Surface epithelium damage

Tear Film Instability

Aqueous Tear Deficiency

Evaporative Dry Eye

Tear hyperosmolarity

What do damaged epi cells do that directly contributes to promoting DES?
Dry Eye Syndrome

What do damaged epi cells do that directly contributes to promoting DES?
They release [ ] that promote and/or facilitate inflammation

Inflammatory release

Surface epithelium damage

Hyperosmolar stress

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity
What do damaged epi cells do that directly contributes to promoting DES?
They release cytokines that promote and/or facilitate inflammation.
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They release cytokines that promote and/or facilitate inflammation.

While a number of cytokines are released, the BCSC emphasizes three.

-- TNF
-- MMP-9
-- IL-1

---
Evaporative Dry Eye

Dry Eye Syndrome

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Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity
Dry Eye Syndrome

While a number of cytokines are released, the BCSC emphasizes three. What are they? --? --? --?

- Inflammatory cytokine release
- Surface epithelium damage
- Hyperosmolar stress

- Aqueous Tear Deficiency
- Tear Film Instability
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Dry Eye Syndrome

What do damaged epi cells do that directly contributes to promoting DES?
They release cytokines that promote and/or facilitate inflammation

What negative effects does MMP-9 have on the ocular surface?
It cleaves epi cells from their BM, and from one another, by disrupting junctional elements

How do these effects manifest clinically, ie, at the slit lamp?
As increased fluorescein staining in the form of punctate epithelial erosions
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Dry Eye Syndrome

Inflammation

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TNF and IL-1 have a variety of effects, but the BCSC dwells on one in particular—which is it?

Promotion of apoptosis among surface epi cells (which also leads to PEE)
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Evaporative Dry Eye

Tear hyperosmolarity

What do damaged epi cells do that directly contributes to promoting DES? They release **cytokines** that promote and/or facilitate inflammation.

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Note that surface epi damage induces cytokine release... While a number of cytokines are released, the BCSC emphasizes three. What are they? --TNF --MMP-9 --IL-1

Inflammatory cytokine release

Surface epithelium damage

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Note that surface epi damage induces cytokine release… **And cytokine release induces surface epi damage.**

Aqueous Tear Deficiency  
Tear Film Instability  
Evaporative Dry Eye  
Tear hyperosmolarity
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Note that surface epi damage induces cytokine release... And cytokine release induces surface epi damage. Thus, a vicious cycle/circle develops in which epi damage leads directly to further epi damage.
Dry Eye Syndrome

Inflammatory cytokine release

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Note: Cytokines play another role in DES pathogenesis, one so important that we’re going to discuss it separately. Stay tuned.

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And cytokine release induces surface epi damage.

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Note: Cytokines play another role in DES pathogenesis, one so important that we’re going to discuss it separately. Stay tuned.
Evaporative Dry Eye Syndrome

In addition to cytokine production, surface-epi damage promotes the expression of a particular ‘adhesion’ molecule—which one?

Inflammatory adhesion molecule?

Surface epithelium damage

Hyperosmolar stress

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity
In addition to cytokine production, surface-epi damage promotes the expression of a particular ‘adhesion’ molecule—which one?

Intercellular adhesion molecule 1 (ICAM-1)
In addition to cytokine production, surface-epi damage promotes the expression of a particular ‘adhesion’ molecule—which one? Intercellular adhesion molecule 1 (ICAM-1)

Increased ICAM-1 expression on two cell types are of particular importance vis a vis DES—which cell types?

Vascular endothelial cells and T-lymphocytes

Because they promote and facilitate T-cell migration to the ocular surface (and lacrimal glands)
In addition to cytokine production, surface-epi damage promotes the expression of a particular ‘adhesion’ molecule—which one? Intercellular adhesion molecule 1 (ICAM-1)

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Intercellular adhesion molecule 1 (ICAM-1)

Increased ICAM-1 expression on two cell types are of particular importance vis a vis DES—which cell types?
Vascular endothelial cells and T-lymphocytes

Why is ICAM-1 expression on these cells particularly important in the pathophysiology of DES?
Because it promotes/facilitates T-cell migration to the ocular surface and lacrimal glands, where they play a central role in the inflammatory response
Now to address that other cytokine effect—what is it?

Dry Eye Syndrome

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity
Dry Eye Syndrome

Now to address that other cytokine effect—what is it?
Impedance of the afferent arm of the LFU reflex arc

Neural reflex arc disruption

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity
Now to address that other cytokine effect—what is it? Impedance of the afferent arm of the LFU reflex arc.

**How does this come about?**

The **BCSC** is vague on this score, stating simply that 'inflammatory cytokines block neural signals for tear secretion.'

---

**Graphical Representation:**

- **Dry Eye Syndrome**
  - **Aqueous Tear Deficiency**
  - **Tear Film Instability**
  - **Evaporative Dry Eye**
  - **Tear hyperosmolarity**
Evaporative Dry Eye

Dry Eye Syndrome

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity

How does this come about?
The BCSC is vague on this score, stating simply that ‘inflammatory cytokines block neural signals for tear secretion’

Impedance of the afferent arm of the LFU reflex arc

Neural reflex arc disruption

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

Now to address that other cytokine effect—what is it?
Dry Eye Syndrome

Diminution of input on the afferent side of the LFU arc leads to what change on the efferent side?

- Neural reflex arc disruption
- Inflammatory cytokine release
- Surface epithelium damage
- Hyperosmolar stress

Aqueous Tear Deficiency → Tear Film Instability → Evaporative Dry Eye

Tear hyperosmolarity
Dry Eye Syndrome

- Decreased aqueous production
- Neural reflex arc disruption
- Inflammatory cytokine release
- Surface epithelium damage
- Hyperosmolar stress

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolality

Diminution of input on the afferent side of the LFU arc leads to what change on the efferent side? Decrease in aqueous production by the lac glands.
Diminution of input on the afferent side of the LFU arc leads to what change on the efferent side? **Decrease** in aqueous production by the lac glands.

**Hol up**—if aqueous production is suppressed, how come so many DES pts present with excessive tearing?

- **Decreased aqueous production**
  - Diminution of input on the afferent side of the LFU arc

**Hyperosmolar stress**
- **Aqueous Tear Deficiency**
- **Tear Film Instability**
- **Evaporative Dry Eye**

**Tear hyperosmolarity**
Evaporative Dry Eye Syndrome

Decreased aqueous production

Diminution of input on the afferent side of the LFU arc leads to what change on the efferent side?

Decrease in aqueous production by the lac glands

Hol up—if aqueous production is suppressed, how come so many DES pts present with excessive tearing?

Early in the DES course there is an inflammation-driven uptick in corneal-nerve activity that increases reflex-driven lacrimal gland stimulation, which produces the oft-observed DES pt c/o of tearing (tl;dr irritated eyes often run water).

Hyperosmolar stress

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity
Dry Eye Syndrome

Decreased aqueous production

Diminution of input on the afferent side of the LFU arc leads to what change on the efferent side?

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Early in the DES course there is an inflammation-driven uptick in corneal-nerve activity that increases reflex-driven lacrimal gland stimulation, which produces the oft-observed DES pt c/o of tearing (tl;dr irritated eyes often run water).

Later in the dz process, cumulative nerve damage leads to a diminution in afferent input and thus a decrease in lac gland stimulation, resulting in the decrease in aqueous production as described here.
Evaporative Dry Eye

Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

Note that neural reflex arc disruption decreases aqueous production…

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity
Evaporative Dry Eye

Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

Note that neural reflex arc disruption decreases aqueous production… And decreased aqueous production worsens tear hyperosmolarity, which in turn starts the entire process over again.

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmosmolarity

DES feedback loop!
Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

Aqueous Tear Deficiency

Tear Film Instability

Evaporative Dry Eye

Tear hyperosmolarity

Note that neural reflex arc disruption decreases aqueous production... \textit{And decreased aqueous production worsens tear hyperosmolarity, which in turn starts the entire process over again.} Thus, a vicious cycle/circle develops in which \textit{decreased aqueous production leads directly to further decreases in aqueous production.}
You may have heard previously of the ‘vicious circle’ of DES. But we have IDed two such locations in the process. So which of these represents the vicious circle?
You may have heard previously of the ‘vicious circle’ of DES. But we have IDed two such locations in the process. So which of these represents the vicious circle? That depends on who you ask, and making you aware of this dependency is the point of this question.
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You may have heard previously of the ‘vicious circle’ of DES. *But we have IDed two such locations in the process. So which of these represents the vicious circle? That depends on who you ask, and making you aware of this dependency is the point of this question.*

*So when getting pimped re the DES vicious circle concept, be aware your attending might have one or the other in mind, and so be prepared to modify your response accordingly!*

Some *Academy* sources refer to this as ‘the’ vicious cycle of DES…

DES feedback loop!
Dry Eye Syndrome

Decreased aqueous production

Hol up—we also identified this (increased TFI $\rightarrow$ hyperosmolar epi damage $\rightarrow$ decreased number of goblet cells $\rightarrow$ increased TFI) vicious circle. What about it?

Conj

$^\wedge$Surface epithelium damage

Tear Film

Hyperosmolar stress

Aqueous Tear Deficiency

Evaporative Dry Eye

Tear Film Instability

Tear hyperosmosmolarity

DES feedback loop!
Hol up—we also identified this (increased TFI → hyperosmolar epi damage → decreased number of goblet cells → increased TFI) vicious circle. What about it? That one seems to get no love from anyone, so I doubt your attending will have it in mind if/when she mentions the ‘vicious circle of DES’.
(This is a good point in the set to take a break)
Dry Eye Syndrome

- Decreased aqueous production
- Neural reflex arc disruption
- Inflammatory cytokine release
- Surface epithelium damage
- Hyperosmolar stress

Aqueous Tear Deficiency
Tear Film Instability
Evaporative Dry Eye

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Dry Eye Syndrome

- Decreased aqueous production
- Neural reflex arc disruption
- Inflammatory cytokine release
- Surface epithelium damage
- Hyperosmolar stress

1) Increase tear volume

Tear volume

Aqueous Tear

Tear Film

Instability

Evaporative Dry Eye

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Evaporative Dry Eye

Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

Inflammatory cytokine release

Surface epithelium damage

Hyperosmolar stress

1) Increase tear volume
2) Decrease tear evaporation

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1) Increase tear volume

2) Decrease tear evaporation

3) Prevent cytokine release and/or mitigate their effects
Dry Eye Syndrome

Decreased aqueous production

What is the most straightforward means of increasing aqueous volume?

1) Increase tear volume

2) Decrease tear evaporation

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

Tear Film Instability

Evaporative Tear evaporation

Dry Eye
Dry Eye Syndrome

Decreased aqueous production

What is the most straightforward means of increasing aqueous volume? Supplementing the tear lake with artificial tears.

1) Increase tear volume

2) Decrease tear evaporation

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Dry Eye Syndrome

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What is the most straightforward means of increasing aqueous volume? Supplementing the tear lake with artificial tears. The Cornea book says tear substitutes are “the mainstay of treatment for ATD.”

1) Increase tear volume

Tear volume

Tear Film Instability

Evaporative Dry Eye

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Artificial tears

What is the formal name for artificial tears?

What is the most straightforward means of increasing aqueous volume?

1) Increase tear volume

Aqueous Tear

Tear Film Instability

Evaporative Dry Eye

Tear evaporation

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Dry Eye Syndrome

Decreased aqueous production

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1) Increase tear volume
   - Tear Film Instability
   - Tear evaporation
   - Evaporative Dry Eye

Tear volume

What is the formal name for artificial tears? They are ophthalmic demulcents.

What is the most straightforward means of increasing aqueous volume? Supplementing the tear lake with artificial tears. The Cornea book says tear substitutes are “the mainstay of treatment for ATD.”

mitigate their effects

1) Increase tear volume
2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

What is a demulcent? A substance that, when applied, soothes inflamed mucous membranes.

The word demulcent can also refer to the specific molecule that conveys the soothing effect; e.g., ‘ATs contain a demulcent that…’

What are the two most common molecules used as demulcents in ATs? Polyvinyl alcohol, and cellulose derivatives.
Evaporative Dry Eye

Dry Eye Syndrome

Decreased aqueous production

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Tear Film Instability

Artificial tears

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Dry Eye Syndrome

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What is the formal name for artificial tears? They are ophthalmic demulcents.

In general terms, what is a demulcent?

What are the two most common molecules used as demulcents in ATs?

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Dry Eye Syndrome

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- Cellulose derivatives
Dry Eye Syndrome

Decreased aqueous production

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- Tear evaporation
- Neural reflex arc disruption

Tear Film Instability

Surface epithelium damage

Inflammatory cytokine release

Decreased aqueous production
Dry Eye Syndrome

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What is the less-formal name for the active ingredient in an AT preparation? Artificial tears

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Dry Eye Syndrome

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1) Increase tear volume

Mitigate their effects

What is the formal name for artificial tears? They are ophthalmic demulcents.

What is the less-formal name for the active ingredient in an AT preparation? A wetting agent; eg, “Carboxymethylcellulose is the wetting agent in a number of AT formulations.”

The word demulcent can also refer to the specific molecule that conveys the soothing effect; eg, “ATs contain a demulcent that…” What are the two most common molecules used as demulcents in ATs? Polyvinyl alcohol, and cellulose derivatives, eg, methylcellulose.

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Dry Eye Syndrome

Decreased aqueous production

Evaporative

Dry Eye

Aqueous Tear Deficiency

Tear Hyperosmolarity

Hyperosmolar stress

Tear Film Instability

Surface epithelium damage

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What is the most straightforward means of increasing aqueous volume? Supplementation the tear lake with artificial tears. The Cornea book says tear substitutes are “the mainstay of treatment for ATD.”

What is the formal name for artificial tears? They are ophthalmic demulcents.

In general terms, what is a demulcent? A substance that, when applied, soothes inflamed mucous membranes.

The word demulcent can also refer to the specific molecule that conveys the soothing effect; eg, ‘ATs contain a demulcent that…’ What are the two most common molecules used as demulcents in ATs? Polyvinyl alcohol, and cellulose derivatives, eg, methylcellulose.

What is the less-formal name for the active ingredient in an AT preparation? A wetting agent.

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Dry Eye Syndrome

Decreased aqueous production

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Artificial tears

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artificial tears

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Inflammatory cytokine release

Surface epithelium damage

Tear Film Instability

Decompressed Tear Evaporation

Decreased aqueous production

Tear volume

Aqueous Tear Production

Tear Film Hyperosmolarity

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Dry Eye Syndrome

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Artificial tears

Tear film

Instability

Tear evaporation

Dry Eye

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Artificial tears

Dermulcent

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Briefly, what is involved in each?

Reversible and permanent. In reversible occlusion, a plug (composed of silicone, usually) is stuffed into the punctum, blocking it. Permanent occlusion involves applying heat to the inner aspect of the punctum, scarring it closed.
Dry Eye Syndrome

Decreased aqueous production

Evaporative Dry Eye

Dry Eye Syndrome

Aqueous Tear Deficiency

Tear hyperosmolarity

Hyperosmolar stress

Tear Film Instability

Surface epithelium damage

Neural reflex arc disruption

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Dry Eye Syndrome

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1) Increase tear volume

Tear evaporation

Tear Film

Instability

Dry Eye

Aquous Tear

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Dry Eye Syndrome

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1) Increase tear volume

What complications are associated with punctal occlusion?

There are indeed—**punctal occlusion** is a commonly-performed procedure in moderate to severe ATD.

**There are two general ways to occlude the puncta**—what are they?

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**What is the name of the procedure that increases aqueous production?**

**What is the procedure that decreases tear evaporation?**

**What is the procedure that mitigates the release of inflammatory cytokines?**

---

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

- **Tear Volume**
- **Tear Film Instability**
- **Dry Eye**
Dry Eye Syndrome

Decreased aqueous production

What complications are associated with punctal occlusion?
--They can fail: Inserts can be dislodged; adhesions can open up
--?

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Dry Eye Syndrome

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**Dry Eye Syndrome**

**Decreased aqueous production**

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Surface epithelium damage
Neural reflex arc disruption

Inflammatory cytokine release
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- Decreased aqueous production
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Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it?

Lid hygiene. The Cornea book says lid hygiene is “an essential part [of tx] at all stages of the disease.”

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Dry Eye Syndrome

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1) Increase tear volume
2) Decrease tear evaporation
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Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

Hyperosmolar stress

Inflammatory cytokine release

Surface epithelium damage

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Tear Film Instability

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Tear evaporation

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What steps/interventions can be taken in this regard?

--Topical abx
--Topical steroids
--PO tetracyclines changes
--O3FA supplementation

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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
OK, so heat and massage get the abnormal meibum flowing—then what? Won’t the remaining portion just become semisolid again after the lid cools off? Indeed it will—that is, unless steps are taken to normalize its chemical composition.

What steps/interventions can be taken in this regard?

1) Increase tear volume
2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it? Lid hygiene. The Cornea book says lid hygiene is “an essential part [of tx] at all stages of the disease.”

What are the two fundamental steps involved in lid hygiene?

1) Application of heat to the eyelids to soften the abnormal meibum
2) Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is ‘abnormal’?

It means its chemical composition has been altered (and not for the better).

What are the knock-on effects of this chemical abnormality?

There are several, but chief among them is they induce a change in the melting point of meibum, i.e., the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it’s a semisolid means it can’t even get out and onto the tear film.

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What steps/interventions can be taken in this regard?
Dry Eye Syndrome

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--Topical
--Topical
--?
--?

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--- Topical abx
--- Topical steroids
--- PO
--- PO

Lid hygiene

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--Topical abx
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Dry Eye Syndrome

OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just get the remaining meibum clogging for the lid to cool off? Indeed it will—the point of hyperosmolar stress & dry eye syndrome is that it's a semisolid means it can't even get out and onto the tear film.

What steps/interventions can be taken to normalize the chemical composition of meibum?

1) Application of heat to the eyelids to soften the abnormal meibum
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Why is bacterial load diminution helpful?

Bacterial lipases play an important role in altering meibum's chemical composition. Reduce the bacterial load → reduce the lipase load → reduce the rate and degree of meibum alteration.

Which topical abx is preferred for this?

Azithromycin

Lid hygiene is an essential part of tx at all stages of the disease.

What two fundamental steps are involved in lid hygiene?

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Dry Eye Syndrome

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1. **Decrease tear evaporation**
2. **Increase tear volume**
3. **Prevent cytokine release and/or mitigate their effects**

**Lid hygiene**

What two fundamental steps are involved in lid hygiene?

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Topical abx

Why is topical abx preferred for this?

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Why is bacterial load diminution helpful?

Bacterial lipases play an important role in altering meibum’s chemical composition. Reduce the bacterial load → reduce the lipase load → reduce the rate and degree of meibum alteration.

What steps/interventions can be taken in this regard?

--Topical abx
--Topical steroids
--PO tetracyclines
--PO O3FA
--Bacterial load diminution is helpful because bacterial lipases play an important role in altering meibum’s chemical composition. Reduce the bacterial load → reduce the lipase load → reduce the rate and degree of meibum alteration.

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Dry Eye Syndrome

Evaporative Dry Eye

Dry Eye Syndrome

Aqueous Tear Deficiency

Tear hyperosmolarity

Hyperosmolar stress

Tear Film Instability

Surface epithelium damage

Neural reflex arc disruption

Inflammatory cytokine release

Decreased aqueous production

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Which topical abx is preferred for this?

Topical abx: Azithromycin

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Dry Eye Syndrome

OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just be reabsorbed by the glands, just like before? Indeed it will—unless steps are taken to normalize its chemical composition.

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What steps/interventions can be taken in this regard?

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-- Topical steroids
-- PO tetracyclines
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What steps/interventions are possible?
- Topical abx
- Topical steroids
- PO tetracyclines
- PO O3FA

Topical abx in the pt is already on topical azithromycin—isn't that redundant?

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- **Topical abx**
- **PO tetracyclines**
- **PO O3FA**

PO tetracyclines in the pt is already on topical azithromycin— isn’t that redundant? You’d think so, but no—in MGD management, tetracyclines act primarily as an anti-inflammatory.

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What steps/interventions?
- **PO tetracyclines**
- **Topical abx**
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**What two anti-inflammatory properties do they possess?**
-?
-?

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What two anti-inflammatory properties do they possess?
- They reduce cytokine release
- ?

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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Dry Eye Syndrome

OK, so heat and massage get the abnormal meibum flowing—then what? Won’t the remaining portion just become semisolid again after the lid cools off? Indeed it will—that is, unless steps are taken to normalize its chemical composition.

What steps/interventions?
- Topical abx
- Topical steroids
- PO tetracyclines
- PO O3FA

PO tetracyclines in the pt is already on topical azithromycin—isn’t that redundant?
You’d think so, but no—in MGD management, tetracyclines act primarily as an anti-inflammatory.

What two anti-inflammatory properties do they possess?
--They reduce cytokine release
--They reduce inflammation.

So the logic underpinning lid hygiene is:

1) Increase tear volume
--Step 1: Liquify the semisolid abnormal meibum clogging the glands
--Step 2: Express the now-liquefied abnormal meibum from the glands

2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

What does it mean to say meibum is ‘abnormal’?
It means its chemical composition has been altered (and not for the better).

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Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it? **Lid hygiene**. The Cornea book says lid hygiene is “an essential part [of tx] at all stages of the disease.”

What two fundamental steps are involved in lid hygiene?

1. **Application of heat to the eyelids to soften the abnormal meibum**
2. **Compression/massage of the lid margin to express the abnormal meibum**

What does it mean to say meibum is ‘abnormal’?

It means its chemical composition has been altered (and not for the better)

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What two anti-inflammatory properties do they possess?

--They reduce cytokine release
--They inhibit MMP-9 activity

PO tetracyclines

Topical abx

Topical steroids

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Dry Eye Syndrome

Evaporative Dry Eye

Dry Eye Syndrome

Aqueous Tear Deficiency

Tear hyperosmolarity

Hyperosmolar stress

Tear Film Instability

Surface epithelium damage

Neural reflex arc disruption

Inflammatory cytokine release

Decreased aqueous production

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1) Application of heat to the eyelids to soften the abnormal meibum
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What are the knock-on effects of this chemical abnormality? There are several, but chief among them is they induce a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it’s a semisolid means it can’t even get out and onto the tear film.

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OK, so heat and massage get the abnormal meibum flowing—then what? Won’t the remaining portion just become semisolid again after the lid cools off? Indeed it will—but that is where the logic underpinning lid hygiene comes in:

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Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

What steps/interventions can be taken in this regard?

---

PO tetracyclines
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Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

It must be dosed frequently, and it must be taken on an empty stomach

What are the two alternatives that lack these limitations?

Minocycline and doxycycline

How long is a typical course of tx?
4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?
Yes
Evaporative Dry Eye

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Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

It must be dosed frequently

What are the two alternatives that lack these limitations?

Minocycline and doxycycline

How long is a typical course of tx?

4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes
Dry Eye Syndrome

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Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it? Lid hygiene. The Cornea book says lid hygiene is “an essential part [of tx] at all stages of the disease.”

What two fundamental steps are involved in lid hygiene?

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Dry Eye Syndrome

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1) Increase tear volume
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OK, so heat and massage get the abnormal meibum flowing—then what? Won’t the remaining portion hydrolyze back to its original semisolid state? Indeed it will—that is why lid hygiene must be repeated regularly. Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it?

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So the logic underpinning lid hygiene is:

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Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

It must be dosed frequently, and it must be taken on an empty stomach

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
**Dry Eye Syndrome**

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OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off?

Indeed it will—that is, unless steps are taken to normalize its chemical composition

What steps/interventions can be taken in this regard?

- Topical abx
- Topical steroids
- PO tetracyclines
- PO O3FA

PO tetracyclines

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

It must be dosed frequently, and it must be taken on an empty stomach

What are the two alternatives that lack these limitations?

- Topical abx
- Topical steroids
- PO tetracyclines
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Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

It must be dosed frequently, and it must be taken on an empty stomach

What are the two alternatives that lack these limitations?

- Topical abx
- Topical steroids
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- PO O3FA

Minocycline and doxycycline

How long is a typical course of tx?

4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes
With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Dry Eye Syndrome

OK, so heat and massage get the abnormal meibum flowing—then what? Won’t the remaining portion solidify again? Indeed it will—that is why lid hygiene is an ongoing treatment.

What steps/interventions are involved in lid hygiene?

1) Application of heat to the eyelids to soften the abnormal meibum
2) Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is ‘abnormal’?

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So the logic underpinning lid hygiene is:

---Step 1: Liquify the semisolid abnormal meibum clogging the glands
---Step 2: Express the now-liquefied abnormal meibum from the glands

PO tetracyclines

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

It must be dosed frequently, and it must be taken on an empty stomach

What are the two alternatives that lack these limitations?

Minocycline and doxycycline

How long is a typical course of tx?

4-6 weeks, maybe a little longer

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Yes
Dry Eye Syndrome

OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off? Indeed it will—that is why the logic underpinning lid hygiene is:

1) Increase tear volume
2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

What steps/interventions?
--Topical abx
--Topical steroids
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PO tetracyclines

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations? It must be dosed frequently, and it must be taken on an empty stomach.

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--Step 1: Liquify the semisolid abnormal meibum clogging the glands
--Step 2: Express the now-liquefied abnormal meibum from the glands

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Dry Eye Syndrome

OK, so heat and massage get the abnormal meibum flowing—then what? Won’t the remaining portion just solidify again? Indeed it will—that is why prepping the gaze before lid hygiene is important.

What steps/interventions are involved in lid hygiene?
- Topical abx
- Topical steroids
- PO tetracyclines

PO tetracyclines

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?
- It must be dosed frequently, and it must be taken on an empty stomach

What are the two alternatives that lack these limitations?
- Minocycline and doxycycline

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4-6 weeks, maybe a little longer

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- Step 1: Liquify the semisolid abnormal meibum clogging the glands
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Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it?

Lid hygiene. The Cornea book says lid hygiene is “an essential part [of tx] at all stages of the disease.”

What are the two most important steps involved in lid hygiene?

1) Application of heat to the eyelids to soften the abnormal meibum

2) Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is ‘abnormal’?

It means its chemical composition has been altered (and not for the better).

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OK, so heat and massage get the abnormal meibum flowing—then what? Won’t the remaining portion just become semisolid again after the lid cools off? Indeed it will—that is, unless steps are taken to normalize its chemical composition.

**Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?**

It must be dosed frequently, and it must be taken on an empty stomach.

**What are the two alternatives that lack these limitations?**

Minocycline and doxycycline.

How long is a typical course of tx?

4-6 weeks, maybe a little longer.

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes.

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

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Yes.
Dry Eye Syndrome

OK, so heat and massage get the abnormal meibum flowing—then what? Won’t the remaining portion solidify and clog the glands? Indeed it will—that is the point of the heat and massage.

What steps/interventions might be considered?

- Topical abx
- Topical steroids
- PO tetracyclines
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PO tetracyclines

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Dry Eye Syndrome

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

- Topical abx
- Topical steroids
- PO tetracyclines
- PO O3FA

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

So the logic underpinning lid hygiene is:

1) Increase tear volume
   --Step 1: Liquify the semisolid abnormal meibum clogging the glands
   --Step 2: Express the now-liquefied abnormal meibum from the glands

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

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2) Decrease tear evaporation
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Lid hygiene is the mainstay tx of EDE. What is it?

Lid hygiene is an essential part of tx at all stages of the disease.

What are the two fundamental steps involved in lid hygiene?

1) Application of heat to the eyelids to soften the abnormal meibum
2) Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is 'abnormal'?

It means its chemical composition has been altered (and not for the better)

What are the knock-on effects of this chemical abnormality?

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What steps/interventions can be taken in this regard?

--Topical abx
--Topical steroids
--PO tetracyclines

PO tetracyclines

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

--Photosensitization

What are the two alternatives that lack these limitations?

Minocycline and doxycycline

How long is a typical course of tx?

4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

--Photosensitization

--?

--?

--?

--?

--?

Can the tetracyclines be used during pregnancy? Breastfeeding?

No. No.
Dry Eye Syndrome

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

--Photosensitization (pts should be instructed to avoid sun exposure)

What steps/interventions can be taken to normalize the chemical composition of meibum?

--Topical abx
--Topical steroids
--PO tetracyclines
--PO O3FA

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

So the logic underpinning lid hygiene is:

--Step 1: Liquify the semisolid abnormal meibum clogging the glands
--Step 2: Express the now-liquefied abnormal meibum from the glands

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Dry Eye Syndrome

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1. **Increase tear volume**
2. **Decrease tear evaporation**
3. **Prevent cytokine release and/or mitigate their effects**

The Cornea book says lid hygiene is “an essential part [of tx] at all stages of the disease.”

What two fundamental steps are involved in lid hygiene?

1. Application of heat to the eyelids to soften the abnormal meibum
2. Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is ‘abnormal’?

It means its chemical composition has been altered (and not for the better)

What are the knock-on effects of this chemical abnormality?

There are several, but chief among them is they induce a change in the melting point of meibum, meaning the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it's a semisolid means it can't even get out and onto the tear film.

Is it considered appropriate to repeat the course if the initial response was less than hoped-for? Yes

PO tetracyclines

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

--Photosensitization (pts should be instructed to avoid sun exposure)
--GI upset
--?
--?

Topical abx
Topical steroids
PO O3FA
PO tetracyclines

What are some of the significant side effects of tetracyclines?

--Photosensitization (pts should be instructed to avoid sun exposure)
--GI upset
--?
--?

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

It must be dosed frequently, and it must be taken on an empty stomach

What are the two alternatives that lack these limitations?

Minocycline and doxycycline

How long is a typical course of tx?

4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

Can the tetracyclines be used during pregnancy? Breastfeeding?

No. No.
Evaporative Dry Eye

Aqueous Tear Deficiency

Tear hyperosmolarity

Tear Film Instability

Surface epithelium damage

Neural reflex arc disruption

Inflammatory cytokine release

Decreased aqueous production

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1) Increase tear volume
2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it?

Lid hygiene.

The Cornea book says lid hygiene is "an essential part [of tx] at all stages of the disease."

What two fundamental steps are involved in lid hygiene?

1) Application of heat to the eyelids to soften the abnormal meibum
2) Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is 'abnormal'?

It means its chemical composition has been altered (and not for the better)

What are the knock-on effects of this chemical abnormality?

There are several, but chief among them is they induce a change in the melting point of meibum, i.e., the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it's a semisolid means it can't even get out and onto the tear film.

So the logic underpinning lid hygiene is:

1) Step 1: Liquify the semisolid abnormal meibum clogging the glands
2) Step 2: Express the now-liquefied abnormal meibum from the glands

OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off?

Indeed it will—that is, unless steps are taken to normalize its chemical composition

What steps/interventions can be taken in this regard?

--Topical abx
--Topical steroids
--PO tetracyclines
--PO O3FA

PO tetracyclines

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

--Photosensitization (pts should be instructed to avoid sun exposure)
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Topical abx

Topical steroids

PO tetracyclines in the pt is already on topical azithromycin—isn't that redundant?

You'd think so, but no—in MGD management, tetracyclines act primarily as an anti-inflammatory

What two anti-inflammatory properties do they possess?

--They reduce cytokine release
--They inhibit MMP-9 activity

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

It must be dosed frequently, and it must be taken on an empty stomach

What are the two alternatives that lack these limitations?

Minocycline and doxycycline

How long is a typical course of tx?

4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

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Can the tetracyclines be used during pregnancy? Breastfeeding?

No. No.
Dry Eye Syndrome

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1) Increase tear volume
2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it? Lid hygiene. The Cornea book says lid hygiene is “an essential part [of tx] at all stages of the disease.”

What two fundamental steps are involved in lid hygiene?

1) Application of heat to the eyelids to soften the abnormal meibum
2) Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is ‘abnormal’?

It means its chemical composition has been altered (and not for the better)

What are the knock-on effects of this chemical abnormality?

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What steps/interventions can be taken in this regard?

--Topical abx
--Topical steroids
--PO tetracyclines
--PO O3FA

PO tetracyclines

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

--Photosensitization (pts should be instructed to avoid sun exposure)
--GI upset
--Potentiation of effect in certain anticoagulant meds

It must be dosed frequently, and it must be taken on an empty stomach

What are the two alternatives that lack these limitations?

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--Photosensitization (pts should be instructed to avoid sun exposure)
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Can the tetracyclines be used during pregnancy? Breastfeeding?

No. No.
Dry Eye Syndrome

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1. **Increase Tear Volume**
   - Step 1: Liquify the semisolid abnormal meibum clogging the glands
   - Step 2: Express the now-liquefied abnormal meibum from the glands

2. **Decrease Tear Evaporation**

3. **Prevent cytokine release and/or mitigate their effects**

Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it? **Lid hygiene**. The Cornea book says lid hygiene is "an essential part [of tx] at all stages of the disease."

What two fundamental steps are involved in lid hygiene?
1. Application of heat to the eyelids to soften the abnormal meibum
2. Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is 'abnormal'?
It means its chemical composition has been altered (and not for the better)

What are the knock-on effects of this chemical abnormality?
There are several, but chief among them is they induce a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it's a semisolid means it can't even get out and onto the tear film.

So the logic underpinning lid hygiene is:

1) **Increase Tear Volume**
2) **Decrease Tear Evaporation**
3) **Prevent cytokine release and/or mitigate their effects**

OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off?
Indeed it will—that is, unless steps are taken to normalize its chemical composition.

What steps/interventions can be taken in this regard?

- **PO tetracyclines**
- **Topical abx**
- **Topical steroids**
- **PO O3FA**

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?
- Photosensitization (pts should be instructed to avoid sun exposure)
- GI upset
- Potentiation of effect in certain anticoagulant meds (classic example: ?)

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?
Yes

What are some of the significant side effects of tetracyclines?
- Photosensitization (pts should be instructed to avoid sun exposure)
- GI upset
- Potentiation of effect in certain anticoagulant meds (classic example: warfarin)
- Photosensitivity

Can the tetracyclines be used during pregnancy? Breastfeeding?
No. No.
Ok, so heat and massage of the remaining portion of the abnormal meibum—indeed it will—that is the remaining portion of the abnormal meibum.

What steps/interventions can be used to normalize the remaining portion of the abnormal meibum?

- Topical abx
- Topical steroids
- PO tetracyclines
- PO O3FA

PO tetracyclines

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

- Photosensitization (pts should be instructed to avoid sun exposure)
- GI upset
- Potentiation of effect in certain anticoagulant meds (classic example: warfarin)

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

So the logic underpinning lid hygiene is:

1) Increase tear volume
   - Step 1: Liquify the semisolid abnormal meibum clogging the glands
   - Step 2: Express the now-liquefied abnormal meibum from the glands

2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it?

Lid hygiene. The Cornea book says lid hygiene is "an essential part [of tx] at all stages of the disease."

What two fundamental steps are involved in lid hygiene?

1) Application of heat to the eyelids to soften the abnormal meibum
2) Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is 'abnormal'?

It means its chemical composition has been altered (and not for the better)

What are the knock-on effects of this chemical abnormality?

There are several, but chief among them is they induce a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it's a semisolid means it can't even get out and onto the tear film.

So the logic underpinning lid hygiene is:

1) Increase tear volume
   - Step 1: Liquify the semisolid abnormal meibum clogging the glands
   - Step 2: Express the now-liquefied abnormal meibum from the glands

2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off?

Indeed it will—that is, unless steps are taken to normalize its chemical composition.

What steps/interventions can be taken in this regard?

- Topical abx
- Topical steroids
- PO tetracyclines
- PO O3FA

PO tetracyclines

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

- Photosensitization (pts should be instructed to avoid sun exposure)
- GI upset
- Potentiation of effect in certain anticoagulant meds (classic example: warfarin)
- ?
- ?
- ?
- ?

Can the tetracyclines be used during pregnancy? Breastfeeding?

No. No.
Dry Eye Syndrome

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1. **Tear Volume**
   - **Step 1**: Increase tear production
   - **Step 2**: Decrease tear evaporation

2. **Tear Instability**
   - Prevent cytokine release and/or mitigate their effects

3. **Tear Film**
   - Prevent surface epithelium damage
   - Neural reflex arc disruption
   - Inflammatory cytokine release

Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it? **Lid hygiene**. The Cornea book says lid hygiene is “an essential part [of tx] at all stages of the disease.”

What two fundamental steps are involved in lid hygiene?

1. Application of heat to the eyelids to soften the abnormal meibum
2. Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is ‘abnormal’?

It means its chemical composition has been altered (and not for the better)

What are the knock-on effects of this chemical abnormality?

There are several, but chief among them is they induce a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it’s a semisolid means it can’t even get out and onto the tear film.

So the logic underpinning lid hygiene is:

---

1. **Step 1**: Liquify the semisolid abnormal meibum clogging the glands
2. **Step 2**: Express the now-liquefied abnormal meibum from the glands

OK, so heat and massage get the abnormal meibum flowing—then what? Won’t the remaining portion just become semisolid again after the lid cools off? Indeed it will—that is, unless steps are taken to normalize its chemical composition.

What steps/interventions can be taken in this regard?

---

- **Topical abx**
- **Topical steroids**
- **PO tetracyclines**
- **PO O3FA**

PO tetracyclines

Is it considered appropriate to repeat the course if the initial response was less than hoped-for? Yes

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

---

- Photosensitization (pts should be instructed to avoid sun exposure)
- GI upset
- Potentiation of effect in certain anticoagulant meds (classic example: warfarin)
- Reduction in effectiveness of oral contraceptives
- Photosensitization

Topical abx

It must be dosed frequently, and it must be taken on an empty stomach.

What are the two alternatives that lack these limitations?

- Minocycline
- Doxycycline

How long is a typical course of tx?

4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

---

- Photosensitization (pts should be instructed to avoid sun exposure)
- GI upset
- Potentiation of effect in certain anticoagulant meds (classic example: warfarin)
- Reduction in effectiveness of oral contraceptives
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- Reduction in effectiveness of oral contraceptives
- Photosensitization
- GI upset
- Potentiation of effect in certain anticoagulant meds (classic example: warfarin)

Can the tetracyclines be used during pregnancy? Breastfeeding?

No. No.
Evaporative Dry Eye Syndrome

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1) Increase tear volume
2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

What are the knock-on effects of the chemical abnormality in meibum?
- There are several, but chief among them is a change in the melting point of meibum. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it's a semisolid means it can't even get out and onto the tear film.

What steps/interventions can be taken in this regard?
- Topical anti-inflammatory agents
- Topical steroids
- PO tetracyclines
- PO O3FA

PO tetracyclines

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?
Yes

Why is lid hygiene important?
- The cornea book says lid hygiene is “an essential part of tx at all stages of the disease.”

What two fundamental steps are involved in lid hygiene?
1) Application of heat to the eyelids to soften the abnormal meibum
2) Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is ‘abnormal’?
- It means its chemical composition has been altered (and not for the better)

What are the knock-on effects of this chemical abnormality?
- There are several, but chief among them is a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies.

1) Increase tear volume

Why are topical anti-inflammatory agents, particularly tetracyclines, important in the treatment of DES?
- They reduce cytokine release
- They inhibit MMP-9 activity

What are the limitations of tetracycline use?
- It must be dosed frequently
- It must be taken on an empty stomach

What are the two alternatives that lack these limitations?
- Minocycline
- Doxycycline

How long is a typical course of tx?
4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?
Yes

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?
- Photosensitization (pts should be instructed to avoid sun exposure)
- GI upset
- Potentiation of effect in certain anticoagulant meds (classic example: warfarin)
- Reduction in effectiveness of oral contraceptives

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?
- It must be dosed frequently
- It must be taken on an empty stomach

What are some of the significant side effects of tetracyclines?
- Photosensitization (pts should be instructed to avoid sun exposure)
- GI upset
- Potentiation of effect in certain anticoagulant meds (classic example: warfarin)
- Reduction in effectiveness of oral contraceptives

Can the tetracyclines be used during pregnancy? Breastfeeding?
No. No.
Dry Eye Syndrome

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1) Increase tear volume
2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it?

Lid hygiene. The Cornea book says lid hygiene is "an essential part [of tx] at all stages of the disease."

What two fundamental steps are involved in lid hygiene?

1) Application of heat to the eyelids to soften the abnormal meibum
2) Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is 'abnormal'?

It means its chemical composition has been altered (and not for the better)

What are the knock-on effects of this chemical abnormality?

There are several, but chief among them is they induce a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it's a semisolid means it can't even get out and onto the tear film.

So the logic underpinning lid hygiene is:

--Step 1: Liquify the semisolid abnormal meibum clogging the glands
--Step 2: Express the now-liquefied abnormal meibum from the glands

OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off?

Indeed it will—that is, unless steps are taken to normalize its chemical composition

What steps/interventions can be taken in this regard?

--Topical abx
--Topical steroids
--PO tetracyclines
--PO O3FA
--PO tetracyclines in the pt is already on topical azithromycin—isn't that redundant?

You'd think so, but no—in MGD management, tetracyclines act primarily as an anti-inflammatory

What two anti-inflammatory properties do they possess?

--They reduce cytokine release
--They inhibit MMP-9 activity

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

It must be dosed frequently, and it must be taken on an empty stomach

What are the two alternatives that lack these limitations?

Minocycline and doxycycline

How long is a typical course of tx?

4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

--Photosensitization (pts should be instructed to avoid sun exposure)
--GI upset
--Potentiation of effect in certain anticoagulant meds (classic example: warfarin)
--Reduction in effectiveness of oral contraceptives
--Teeth discoloration in children

Can the tetracyclines be used during pregnancy? Breastfeeding?

No. No.

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

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Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

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--Teeth discoloration in children

Can the tetracyclines be used during pregnancy? Breastfeeding?

No. No.
Dry Eye Syndrome

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1) Increase tear volume
2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

What steps/interventions?

- **PO tetracyclines**
- Topical abx
- Topical steroids
- PO O3FA
- Lid hygiene

What does it mean to say meibum is 'abnormal'?

It means its chemical composition has been altered (and not for the better).

What are the knock-on effects of this chemical abnormality?

There are several, but chief among them is they induce a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it's a semisolid means it can't even get out and onto the tear film.

So the logic underpinning lid hygiene is:

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**Step 1**: Liquify the semisolid abnormal meibum clogging the glands
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What steps/interventions can be taken in this regard?

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- Topical abx
- Topical steroids
- PO tetracyclines
- PO O3FA

Topical tetracyclines are already on topical azithromycin—isn't that redundant? You’d think so, but no—in MGD management, tetracyclines act primarily as an anti-inflammatory.

What two anti-inflammatory properties do they possess?

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- They reduce cytokine release
- They inhibit MMP-9 activity

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

It must be dosed frequently, and it must be taken on an empty stomach.

What are the two alternatives that lack these limitations?

- Minocycline and doxycycline

How long is a typical course of tx?

4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

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- Photosensitization (pts should be instructed to avoid sun exposure)
- GI upset
- Potentiation of effect in certain anticoagulant meds (classic example: warfarin)
- Reduction in effectiveness of oral contraceptives
- Teeth discoloration in children

Can the tetracyclines be used during pregnancy? Breastfeeding?

No. No.
Dry Eye Syndrome

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1) Increase tear volume
2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

What two fundamental steps are involved in lid hygiene?

1) Application of heat to the eyelids to soften the abnormal meibum
2) Compression/massage of the lid margin to express the abnormal meibum

What does it mean to say meibum is 'abnormal'?

It means its chemical composition has been altered (and not for the better)

What are the knock-on effects of this chemical abnormality?

There are several, but chief among them is they induce a change in the melting point of meibum, ie, the temperature at which the normally liquid meibum solidifies. Normal meibum is a liquid at body temperature, which is why expressed normal meibum looks like tiny drops of vegetable oil. In contrast, the chemically-altered meibum in MGD is a semisolid at body temperature, which is why expressed abnormal meibum looks like toothpaste. So not only is the meibum in MGD altered (and thus less effective), the fact that it's a semisolid means it can't even get out and onto the tear film.

So the logic underpinning lid hygiene is:

---Step 1: Liquify the semisolid abnormal meibum clogging the glands
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OK, so heat and massage get the abnormal meibum flowing—then what? Won't the remaining portion just become semisolid again after the lid cools off?

Indeed it will—that is, unless steps are taken to normalize its chemical composition

What steps/interventions can be taken in this regard?

--Topical abx
--Topical steroids
--PO tetracyclines
--PO O3FA

PO tetracyclines

Can the tetracyclines be used during pregnancy?

No.

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes.

Unlike the limitations above, there are a number of other side effects that are common to all tetracyclines—what are some of the significant ones?

--Photosensitization (pts should be instructed to avoid sun exposure)
--GI upset
--Potentiation of effect in certain anticoagulant meds (classic example: warfarin)
--Reduction in effectiveness of oral contraceptives
--Teeth discoloration in children

What are the two alternatives that lack these limitations?

Minocycline and doxycycline

How long is a typical course of tx?

4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

Yes

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Can the tetracyclines be used during pregnancy?

No.
Evaporative Dry Eye

Dry Eye Syndrome

Aqueous Tear Deficiency

Tear hyperosmolarity

Hyperosmolar stress

Tear Film Instability

Surface epithelium damage

Neural reflex arc disruption

Inflammatory cytokine release

Decreased aqueous production

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1) Increase tear volume
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3) Prevent cytokine release and/or mitigate their effects

Just as ATs are the mainstay of treating ATD, so too is there a mainstay tx of EDE. What is it? Lid hygiene. The Cornea book says lid hygiene is "an essential part [of tx] at all stages of the disease."

What two fundamental steps are involved in lid hygiene?

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Topical abx

Topical steroids

PO tetracyclines

PO O3FA

Can the tetracyclines be used during pregnancy? No

Is it considered appropriate to repeat the course if the initial response was less than hoped-for? Yes

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--Teeth discoloration in children

Tetracycline use has limitations that make alternative cyclines easier to use. What are these limitations?

It must be dosed frequently, and it must be taken on an empty stomach

What are the two alternatives that lack these limitations?

Minocycline and doxycycline

How long is a typical course of tx?

4-6 weeks, maybe a little longer

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?

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Yes
Dry Eye Syndrome

1) Increase tear volume
   --Step 1: Liquify the semisolid abnormal meibum clogging the glands
   --Step 2: Express the now-liquefied abnormal meibum from the glands

2) Decrease tear evaporation
   --Topical abx
   --Topical steroids
   --PO tetracyclines (PO O3FA)

3) Prevent cytokine release and/or mitigate their effects
   --Topical abx
   --Topical steroids
   --PO tetracyclines

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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1. Tear volume
2. Tear evaporation
3. Tear film instability
Dry Eye Syndrome

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--Topical abx
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PO tetracyclines

Can the tetracyclines be used during pregnancy? Breastfeeding?
No. No.

Is it considered appropriate to repeat the course if the initial response was less than hoped-for?
Yes

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Yes
Dry Eye Syndrome

Decreased aqueous production

Neural reflex arc disruption

3) Prevent cytokine release and/or mitigate their effects

Inflammatory cytokine release

What class of topical med is most effective in controlling ocular-surface inflammation?

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:
Dry Eye Syndrome

- Decreased aqueous production
- Neural reflex arc disruption

3) **Prevent cytokine release and/or mitigate their effects**

Inflammatory cytokine release

What class of topical med is most effective in controlling ocular-surface inflammation?
Steroids

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So why don’t we keep all DES pts on them?

Because of their terrible side-effect profile, ie, development of cataracts, increased IOP, and compromised ocular-surface immunity

So steroids are verboten in the management of DES?
Not at all—it’s just that they must be used judiciously (more shortly)
Dry Eye Syndrome

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Tear

Decreased aqueous production

Neural reflex arc disruption

Tear film instability

Surface epithelium damage

Inflammatory cytokine release

Hyperosmolar stress

Tear hyperosmolarity

Aqueous tear deficiency
With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

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--Cyclosporine
--Lifitegrast

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3) **Prevent cytokine release and/or mitigate their effects**

How do they work (in broad terms—not specific mechanisms of action)?
They interfere with the action of T-cells (the recruitment of which is an important cytokine effect)

What’s the main drawback to their use?
It can take a l-o-n-g time for their effects to kick in—weeks (if you’re lucky) to months (probably more typical). During the ramp-up period, compliance may become an issue as the pt gives up in frustration.
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What’s the main drawback to their use?
It can take a l-o-n-g time for their effects to kick in—weeks (if you’re lucky) to months (probably more realistic). During this ramp-up period, the pt may experience worse symptoms as the pt gives up in frustration.

Why does it take so long for these drugs to reach full effect?
It’s probably related to the length of the T-cell life cycle (~120 days)

What can be done to bridge the gap between commencement of therapy and onset of symptom relief?
A short course of topical steroids is ideal for this

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1) Increase tear volume
2) Decrease tear evaporation
3) Prevent cytokine release and/or mitigate their effects

What class of topical med is most effective in controlling ocular-surface inflammation?
Steroids

So why don’t we keep all DES pts on them?
Because of their terrible side-effect profile, ie, development of cataracts, increased IOP, and compromised ocular-surface immunity

So steroids are verboten in the management of DES?
Not at all—it’s just that they must be used judiciously (more shortly)
Dry Eye Syndrome (DES) is characterized by aqueous tear deficiency, which leads to tear film instability and surface epithelium damage. Hyperosmolarity results from evaporation, which disrupts the neural reflex arc and triggers an inflammatory cytokine release.

With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1. **Tear volume**
2. **Tear evaporation**
3. **Prevent cytokine release and/or mitigate their effects**

Two steroid-sparing topical anti-inflammatories are used (in the US). What are they?
- Cyclosporine
- Lifitegrast

How do they work (in broad terms—not specific mechanisms of action)?
They interfere with the action of T-cells (the recruitment of which is an important cytokine effect).

What’s the main drawback to their use?

**It can take a l-o-n-g time for their effects to kick in**—weeks (if you’re lucky) to months (probably more typically). Ensuring the patient remains in the treatment is key, as they may give up before the effects are evident.

Why does it take so long for these drugs to reach full effect? It’s probably related to the length of the T-cell life cycle (~120 days).

What can be done to bridge the gap between commencement of therapy and onset of symptom relief? A short course of topical steroids is ideal for this.

So why don’t we keep all DES pts on them? Because of their terrible side-effect profile, i.e., development of cataracts, increased IOP, and compromised ocular-surface immunity.

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Evaporative Dry Eye

Dry Eye Syndrome

Aqueous Tear Deficiency

Tear hyperosmolarity

Hyperosmolar stress

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It can take a l-o-n-g time for their effects to kick in—weeks (if you’re lucky) to months (probably more typical). Even then, treatment is incomplete, with the pt giving up in frustration as the effects wane.

Why does it take so long for these drugs to reach full effect?

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What’s the main drawback to their use?

It can take a l-o-n-g time for their effects to kick in—weeks (if you’re lucky) to months (probably more typically).

Frustration can run high; patients may be more likely to discontinue treatment due to the lag between initiation of therapy and symptom relief as the pt gives up in frustration.

Why does it take so long for these drugs to reach full effect?

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Two steroid sparing--

Cyclosporine

Lifitegrast

How do they work

They interfere with

What’s the main drawback to their use?

It can take a l-o-n-g time for their effects to kick in—weeks (if you’re lucky) to months (probably more typical). Early adherence is critically important, or the pt may give up in frustration.

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aqueous-tear production

What potential ocular side effects are concerning? Systemic?

It has none (other than stinging). It has no systemic side effects.
**Evaporative Dry Eye**

**Dry Eye Syndrome**

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Tear hyperosmolarity

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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

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Cyclosporine has three measurable effects on the ocular surface—what are they?

- Reduced T-cell numbers

What's the main drawback to their use?

It can take a long time for their effects to kick in—weeks (if you're lucky) to months (probably more realistic). For this reason, patients may become frustrated as the pt gives up in frustration.

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Cyclosporine has three measurable effects on the ocular surface—what are they?

- Reduced T-cell numbers
- Increased conjunctival goblet cell numbers
- (two words)

What potential ocular side effects are concerning? Systemic?

It has none (other than stinging). It has no systemic side effects.
Evaporative Dry Eye

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Cyclosporine has three measurable effects on the ocular surface—what are they?

--Reduced T-cell numbers
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What’s the main drawback to their use?

It can take a long time for their effects to kick in—weeks (if you’re lucky) to months (probably more typical). For a situation in which instant gratification is expected, it can be frustrating to the pt as the pt gives up in frustration.

Why does it take so long for these drugs to reach full effect?

It’s probably related to the length of the T-cell life cycle (~120 days)

What can be done to bridge the gap between commencement of therapy and onset of symptom relief?

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How do they work?

They interfere with the action of T-cells (the recruitment of which is an important cytokine effect)

What’s the main drawback to their use?

It can take a lo-n-g time for their effects to kick in—weeks (if you’re lucky) to months (probably more typical). For instance, it might take 3-6 months for the patient to feel some beneficial effect as the pt gives up in frustration.

2-3 weeks

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--Increased conj goblet cell numbers
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What potential ocular side effects are concerning? Systemic?

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Dry Eye Syndrome

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Tear Film Instability

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Two steroid sparing:

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How do they work?
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Two steroid sparing—

--Cyclosporine
--Lifitegrast

How do they work They interfere with

What’s the main drawback to their use? It can take a l-o-n-g time for their effects to kick in—weeks (if you’re lucky) to months (probably more typical). During this time of inaction, the pt may lose hope as the pt gives up in frustration.

So why don’t we keep all DES pts on them? Because of their terrible side-effect profile, ie, development of cataracts, increased IOP, and compromised ocular-surface immunity

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What potential ocular side effects are concerning? Systemic? It has none (other than stinging). It has no systemic side effects.

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How do they work? They interfere with the action of T-cells (the recruitment of which is an important cytokine effect)

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Lifitegrast

How does lifitegrast reduce T-cell activity?

Cyclosporine has three measurable effects on the ocular surface—

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--Increased conjunctival goblet cell numbers
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What potential ocular side effects are concerning? Systemic?

It has none (other than stinging). It has no systemic side effects.

How does lifitegrast reduce T-cell activity?

By inhibiting ICAM-1 binding

We covered it earlier in the slide-set, but remind me:

What role does ICAM-1 play in the pathophysiology of DES?

It promotes/facilitates T-cell migration to the ocular surface and lacrimal gland
Evaporative Dry Eye

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It can take a looooong time for their effects to kick in—weeks (if you’re lucky) to months (probably more typical). During the ramp-up period, compliance may become an issue as the pt gives up in frustration.

Why does it take so long for these drugs to reach full effect?

It’s probably related to the length of the T-cell life cycle (~120 days)

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Not at all—it’s just that they must be used judiciously (more shortly)

How do they work (in broad terms—not specific mechanisms of action)?

They interfere with the action of T-cells (the recruitment of which is an important cytokine effect)

What’s the main drawback to their use?

It can take a long time for their effects to kick in—weeks (if you’re lucky) to months (probably more typical). During the ramp-up period, compliance may become an issue as the pt gives up in frustration.

Why does it take so long for these drugs to reach full effect?

It’s probably related to the length of the T-cell life cycle (~120 days)

What role does ICAM-1 play in the pathophysiology of DES?

It promotes/facilitates T-cell migration to the ocular surface and lacrimal gland

How does lifitegrast reduce T-cell activity?

By inhibiting ICAM-1 binding

We covered it earlier in the slide-set, but remind me:

Cyclosporine has three measurable effects on the ocular surface—

--Reduced T-cell numbers
--Increased conjunctival goblet cell numbers
--Increased aqueous-tear production

What potential ocular side effects are concerning? Systemic?

It has none (other than stinging). It has no systemic side effects.

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With regards to treating DES—there are three obvious interdiction points in its pathogenesis:

1. Increase tear volume
2. Decrease tear evaporation
3. Prevent cytokine release and/or mitigate their effects

What class of topical med is most effective in controlling ocular-surface inflammation?

Steroids

So why don’t we keep all DES pts on them?

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What can be done to bridge the gap between commencement of therapy and onset of symptom relief?

A short course of topical steroids is ideal for this

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- Aqueous Tear Deficiency
- Tear Film Instability
- Evaporative Dry Eye

Tear

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Most interventions (ATs, anti-inflammatory meds, O3FA) are useful in both conditions. However, there is one relatively common ATD intervention that must be used with caution in pts who also have MGD.

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Why must punctal occlusion be used with caution in ATD pts with concurrent MGD? Because in addition to increasing the amount of aqueous on the ocular surface (good), occlusion will also increase/maintain the proinflammatory abnormal meibum on the ocular surface (bad).
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Why must punctal occlusion be used with caution in ATD pts with concurrent MGD? Because in addition to increasing the amount of aqueous on the ocular surface (good), occlusion will also increase/maintain the proinflammatory abnormal meibum on the ocular surface (bad). In general, you want to control the inflammatory component of a pt’s DES before you occlude their puncta.

Note: There is another complication induced by the use of punctal occlusion that we will cover later in the slide-set.
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Remember when we said this? This is what we were referring to.

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Dry Eye Syndrome

50 Ways to Take a Break

(This is a good point in the set to take a break)
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--Nighttime lagophthalmos
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--- Conjunctivochalasis
--- SLK

Briefly, what is conj’chalasis?

Loose, redundant, nonedematous conj. It usually manifests as a ‘fold’ of conj. draping on the lower-lid margin.

What is the cause?

Probably the mechanical trauma of the lids rubbing against the bulbar conj. during blinking.

What do conj’chalasis pts c/o about?

The same things DES pts do: FBS, red eyes, and tearing.

What is going on, ie, what happens that produces their discomfort?

The redundant conj. chafes against itself during blinking/eye movements.
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How is conj’chalasis managed? It’s reasonable to start with ATs, antihistamines, steroids etc (although one of the characteristics of conj’chalasis is that it doesn’t respond well to DES-tx maneuvers). Often, surgical intervention (in the form of thermal cicatrization or excision) to remove the redundant conj is required for resolution.
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In a nutshell, what is SLK?
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*In a nutshell, what is SLK?*

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Is there a gender predilection?

Yes, females are far more likely to be affected

What do SLK pts c/o?

The same things DES pts do: FBS, red eyes, and tearing

SLK has three classic findings associated with the superior bulbar conj.

What are they?

--Injection

--It is redundant/loose

--It stains with rose bengal, lissamine green, and/or fluorescein

SLK also has a classic tarsal conj finding—what is it?

Papillary reaction

SLK has two classic cornea findings—what are they?

--Superior PEE/K

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Superior rose bengal staining

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--- Conjunctivochalasis

--- SLK

--- In a nutshell, what is SLK?

--- A chronic/recurrent inflammatory condition of the superior limbal cornea and adjacent conj

--- Is it common, or rare?

--- Rare

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K staining pattern in SLK
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What are the two overarching goals in treating SLK?
--?
--?

There are a number of medical treatment options. These include:
--Preservative-free ATs
--Topical anti-inflammatory meds
--Large (enough to cover the involved conj) diameter BCL

Is surgery ever indicated to resolve the redundant conj?
It is indeed
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-- Reduce…surface inflammation
-- Reduce…friction between the superior bulbar conj and superior tarsal conjunctiva

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-- Topical anti-inflammatory medications
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For more on SLK, see slide-set K7
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--Conjunctivochalasis
--SLK
--Floppy eyelid syndrome
--Nighttime lagophthalmos
--Parkinson’s
--Mucous-membrane pemphigoid/OCP

In a nutshell, what is floppy eyelid syndrome (FES)?

A condition characterized by 1) upper-lid laxity and 2) chronic inflammation of the ocular surface.

What do FES pts complain of?

FBs and mucous discharge that are worse in the morning.

What is the presumed pathogenic process in FES?

During sleep, the upper lids evert in response to face-rubbing against a pillow while sleeping in the prone position. Lid eversion results in contact between the eye and the bedding, and this contact traumatizes the ocular epithelia.

What is the main risk factor for FES? (It's systemic, not ocular.)

Obesity

How is FES managed initially?

--Apply ointment to the involved eye(s) at qHS,
--Prevent eversion by either shielding the eye(s) or taping it/them shut

If FES fails to respond to the above, what's next?

Surgical tightening of the lax upper lid(s)

With what potentially lethal systemic condition is FES strongly associated?

Obstructive sleep apnea. The BCSC states that all FES pts should be evaluated for OSA.
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Dry Eye Syndrome

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How can you tell if the UL is lax?

Floppy eyelid syndrome

How can you tell if the UL is lax?
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*How can you tell if the UL is lax?*
It will evert easily and dramatically with traction

**Floppy eyelid syndrome**

How can you tell if the UL is lax?
It will evert easily and dramatically with traction
Dry Eye Syndrome

FES. Wow.
If you can’t tell, that’s an upper lid so lax it can be pinched like this.
Dry Eye Syndrome

FES. Note the fine papillary reaction (another common finding)
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Floppy eyelid syndrome
Dry Eye Syndrome

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